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PATHOLOGY
HARD TISSUES
OF THE TEETH

ORAL DIAGNOSIS

BLACK



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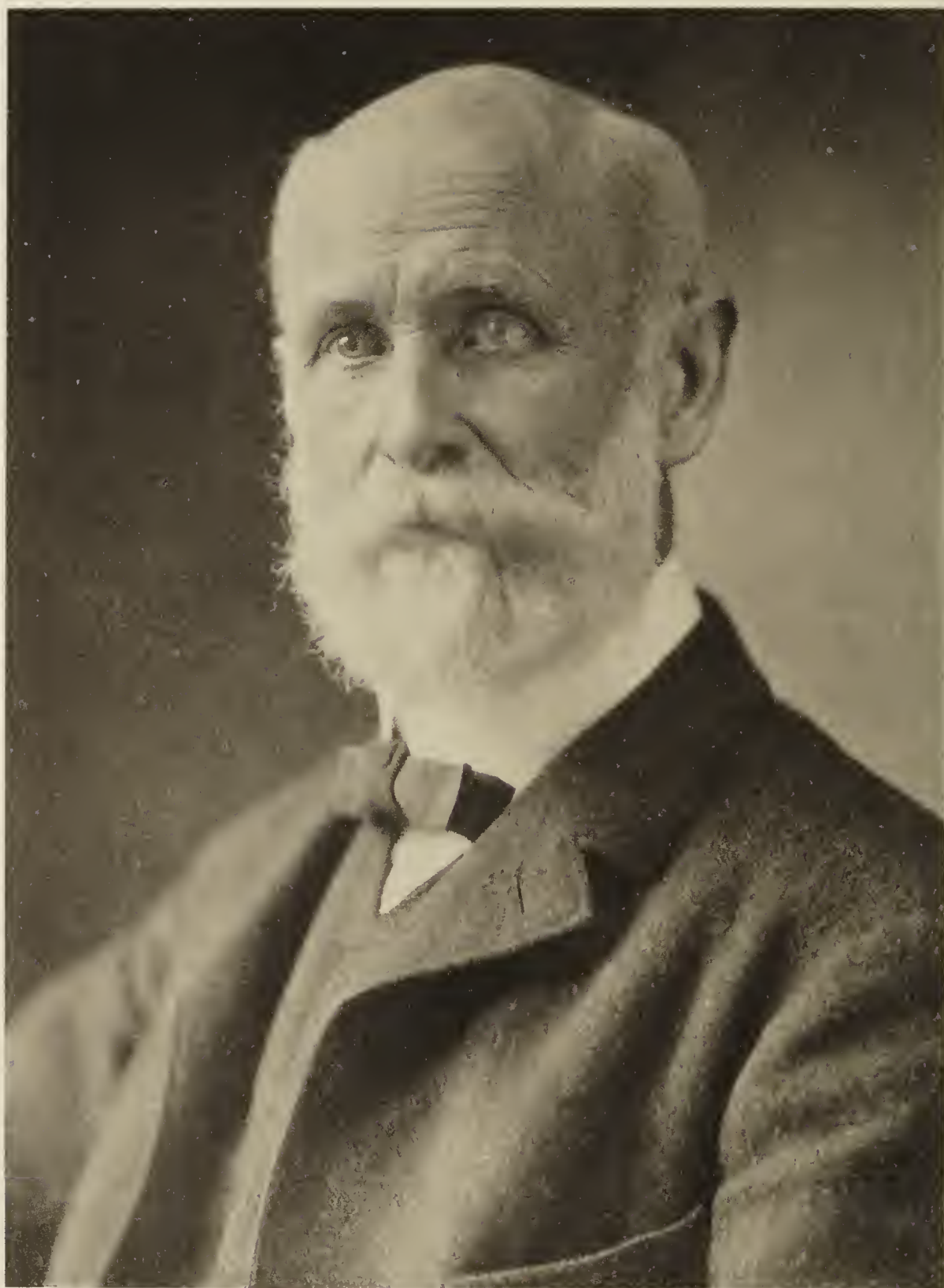
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To Mr. Wilfred Fish
with compliments &

Arthur Beach

December 1936.



Yours truly
G. W. Black

On the One Hundredth Anniversary of his Birth
the Revision of these Volumes
is Dedicated to the Memory of
my Father
Greene Vardiman Black
who, in Devoting his Life to Research,
Advanced Scientific Methods in Dentistry
and Furthered the Conservation of the Teeth

G. V. BLACK'S WORK
ON
OPERATIVE DENTISTRY

with which his
SPECIAL DENTAL PATHOLOGY
IS COMBINED

PUBLISHED IN FOUR VOLUMES

VOLUME ONE
PATHOLOGY OF THE HARD TISSUES
OF THE TEETH
ORAL DIAGNOSIS

280 ILLUSTRATIONS

REVISION BY
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NORTHWESTERN UNIVERSITY DENTAL SCHOOL

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PREFACE

In preparing these volumes, the report of the Curriculum Survey Committee of the American Association of Dental Schools was carefully studied. The subcommittee reports on subjects within or related to the field of operative dentistry were reviewed and the effort was made to harmonize this work with the reports on Histology and Embryology, Oral Anatomy, Nutrition, Mouth Hygiene and Oral Prophylaxis, Application of Preventive Principles, Radiography, Diagnosis and Treatment Planning, Oral Medicine and Oral Surgery. A number of changes suggested by the Subcommittee on Operative Dentistry were made in the nomenclature, the more important of which are mentioned in the Introduction.

Five chapters by other authors are included: Health, Nutrition and Hygiene by Clara M. Davis, M.D., Mottled Enamel by Frederick S. McKay, D.D.S., The Saliva by Harold L. Hansen, Ph.D., and Earl A. Zaus, M.D., Materials Used in Operative Dentistry by Eugene W. Skinner, Ph.D., and Mouth Infection in Relation to Systemic Diseases by Edward H. Hatton, M.D. Each of these chapters is a report of research in a special field over a period of years and the contributors are so familiar with the text as a whole that their writings are directly related to and in fact an integral part of it. Special reference is made to these chapters in the Introduction and the author is grateful for the splendid cooperation of these confreres.

Dr. Hatton, in addition to writing the special chapter just mentioned, contributed much of the data for the chapter on Oral Diagnosis, including the sections on diseases of the soft tissues of the mouth, and the mouth manifestations of systemic diseases and skin diseases, also the review of recent research on dental caries.

Dr. Robert E. Blackwell and Dr. G. R. Lundquist, who have been the Author's associates in the teaching of operative dentistry and oral pathology respectively for many years, have made valuable contributions and have assisted in many other ways in the preparation of this work.

George B. Denton, Ph.D. prepared a very exhaustive review of the literature covering the pathology and treatment of the diseases of the dental pulp and the supporting structures of the teeth, from which much of the historical data was taken.

Dr. William G. Skillen assisted in the preparation of the chapter on the development of the teeth and prepared a large number of photomicrographs illustrating the pathology of the pulp and peridental membranes.

Dr. Charles W. Freeman contributed greatly by direct assistance, particularly in the preparation of the section on oral surgery in the chapter on diagnosis, also by assuming many of the author's

administrative duties at Northwestern University Dental School during the past two years.

Dr. George W. Teuscher gave valuable aid in the preparation of the chapter on the treatment of the temporary teeth.

Substantial assistance has been given by many teachers of operative dentistry who were kind enough to make a special review of the previous edition and offer suggestions for its improvement as a teaching medium. The author is pleased to express his thanks to Drs. H. E. Friesell, Ralph W. Edwards, Louis J. Fitzpatrick, Harry A. True, W. L. Wylie, A. C. LaTouche, Ralph R. Byrnes, Alfred Enloe, J. Ross Tucker, P. V. McParland, C. W. Hagan, E. G. Meisel and C. E. Friesell.

Many acknowledgments are made in the text to other teachers and practitioners who have carried on some related research, reported unusual cases, supplied models, or made valuable suggestions. Among these are Leroy L. Hartman, James M. Prime, J. R. Blayney, Abram Hoffman, B. E. Lischer, Emil Mueller, James R. Schumaker, Leonard S. Fosdick, Ralph G. Bengston, Eugene Bodmer, Arne Foster Romnes, Stanley W. Clark, Roscoe H. Volland, Edgar W. Swanson, Loren D. Sayre, Karl L. Vehe and Lon W. Morrey.

Dr. Robert R. Fosket, Dr. Byron J. May and Miss Helen Snyder supplied many excellent radiographs from their collections, as well as films of especially selected cases. Dr. Roy W. Carr prepared plaster models and Mrs. Dorothy Lee Scantlin did the photographic work for the new illustrations.

Assistance in proof reading was given by Misses Margaret Leach and Dorothy Warren, by Drs. Blackwell, Hatton, Freeman and Swanson, Mr. Gerald VanDoren and Miss Helen Baldwin, also by Mrs. Black, our daughters, Mrs. Barbara Dunbar and Mrs. Jane VanDoren, and our son Gilmer V. Black, who also made a number of drawings.

Acknowledgment is made to The S. S. White Dental Manufacturing Co., the C. L. Frame Dental Supply Co. and the Blue Island Dental Laboratories for their cooperation.

To all of the above the author wishes to express his thanks and appreciation.

This preface is dated on the one hundredth anniversary of my father's birthday.

ARTHUR D. BLACK

Chicago, August 3, 1936.

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OPERATIVE DENTISTRY

Introduction

OPERATIVE DENTISTRY, in the author's conception, consists of all procedures, including preventive measures, by which the teeth may be conserved, and thus maintain the natural masticating mechanism in such a state that the general health will not be endangered.

This necessarily includes the consideration of the etiology and pathology of the diseases of the teeth and their supporting structures, and the title "Special Dental Pathology and Treatment" would be equally appropriate for this work, except for the fact that the care of those cases in which teeth can not be conserved has been, as a matter of convenience, assigned to other departments in teaching, and in a few instances to specialists in practice. The term "Oral Pathology" would include diseases which are not in the strictly dental field.

A strictly dental disease is one that is peculiar to the teeth or their membranes, either in its causation, its nature, or in the tissues to which it is confined, and which does not occur elsewhere in the body. The tissues of the teeth are, in their histology and physiology, a distinct class. The membranes investing the teeth have peculiar histological and physical characters and forms suited to the functions of the teeth. These form a special assemblage of tissues, the pathology of which is unlike that of any other tissues of the body. It is this *special pathology*, together with the manipulation required in treatment, which has made dentistry a *specialty* in medicine.

The present work is devoted primarily to the diseases and treatment (1) of the hard structures of the teeth — caries, hypoplasia, mottled enamel, erosion and abrasion; (2) of the gingivæ and peridental tissues; and (3) of the dental pulp and periapical tissues. The principal reason for presenting these conditions in a single work, lies in the almost universal interrelation of these three groups of diseases and their treatment. In both pathology and treatment, one may stand in direct causal relation to the others; in fact, any one may be related to the other two as cause or effect. One can hardly think of a more complete interrelation of pathological conditions.

In the early days of dental practice, little attention was paid to any of the diseases of these groups as *pathological processes*; they were *conditions* with which the dentist was confronted and

he had at hand several types of mechanical treatment. He filled a cavity, removed a pulp, or possibly only opened into the canal and placed a "peg" crown, or he "pulled" the tooth and replaced it in the manner that seemed most practical. Oftentimes the technical procedure created more disease than it corrected.

It has taken many years for dentistry to evolve from a mechanical pursuit to a scientific branch of the healing art and become interested in the etiology and pathology of disease as a basis for treatment. That this has been in a large measure accomplished, is evidenced by the tremendous advance in preventive measures, which are necessarily based on something of an understanding of pathology.

In view of the fact that the initial lesions of all of the three groups of diseases considered in this work are well understood and are in large measure subject to preventive or immunizing treatment, also in view of the fact that practically all of the other diseases of the dental structures are extensions of these conditions, it seems most logical to include all preventive practices as a part of Operative Dentistry.

CONSERVATION OF THE TEETH.

This work has been prepared on the proposition that the teeth of practically all persons can be conserved throughout life with the means and methods of treatment now available, if the care of the individual is begun early and there is reasonable cooperation between patient and dentist. That this is in large measure an accomplished fact is shown by the number of young adults, twenty-five years of age, or more, many of whom were continually highly susceptible to caries during childhood, who have all of their teeth with vital pulps. They have passed the most critical period and may now be reasonably assured that they will not require dentures later on.

A century ago, in 1835, William Robertson, an English dentist, published a treatise of 158 pages entitled: "Diseases of the Teeth, in which the Origin and Nature of Decay are Explained; and the Means of Prevention Pointed Out." Robertson was the first to classify the positions of beginning decays in the enamel and he led the attack on the then generally accepted theory that decay began within the tooth and progressed outward to the surface. The first paragraph of Robertson's book reads as follows: "The object of the present work is to make the general reader acquainted with the insidious nature of the diseases to which the teeth are liable, to explain to him their causes and progress, and to shew that a knowledge of these is absolutely necessary, either, by early attention, entirely to prevent their occurrence, or by timely

application, to counteract the mischief before it has become irremediable."

This statement evidently expressed Robertson's belief that "early attention" and "timely application" would preserve the teeth. He did not realize that it was to require nearly a century for the dental profession to effectively develop and utilize the plan which he proposed; nor did he appreciate that it would require at least as long to educate a sufficient percentage of the public to an understanding of the possibilities of good dental care.

In an address before the Chicago Dental Society in 1913, Dr. Charles H. Mayo* made the following statement regarding the prevention of disease: "The difference between the knowledge of the layman and the medical attendant, including the dentist, should not be too great. Medical progress may be stayed from time to time that the layman may be educated to certain truths of health; that he must first know, then desire, and then demand proper health conditions."

This statement presents the basis of progress in preventive practice and carries with it certain implications which should be recognized by the professional man as well as the layman, because they are necessary to the successful application of any preventive measure. Certain economic readjustments occur automatically in professional service which not only meet the new situation, but actually work out to the financial advantage of both layman and dentist or physician. The effective application of preventive service involves not only the knowledge by the physician that the method is reliable; but also the education of a large number of laymen to a sufficient understanding of the effectiveness of the treatment, that they will seek the services of the doctor for this purpose. It would be of no particular advantage for every doctor to know that a certain disease could be prevented, provided no layman ever heard of the preventive measure, until he was sick with the disease. Preventive service is dependent upon the spreading of knowledge which causes many persons to demand it. The result is that the physician performs a trivial service, as vaccination, for large numbers during a year, instead of treating a comparatively few persons afflicted with the disease. The many small fees received by the members of the medical profession each year for vaccination to prevent small-pox, must be many times the total fees that were ever collected in one year for treating persons who had contracted this disease.

As preventive measures come to occupy a larger share of the dentist's time he will serve a larger number of persons at lesser fees per person. The service will be less arduous for him and less

* Dental Review, Vol. 27, 1913, p. 281.

painful for his patients, whose health will be better conserved, and this will be to the financial advantage of both.

In 1865, Nathan C. Keep, first president of the Massachusetts State Dental Society, made the following statement in his address: "Under the most favorable conditions, no one should require artificial teeth before the age of thirty-five." Dr. Keep was a leading practitioner of his day; he was the first Dean of Harvard Dental School in 1867, and evidently expressed the hopeful outlook of the profession of that day. Dentistry has made much real progress toward prevention since that time.

Possibly the greatest need today is the recognition, by a larger number of the profession, of the earliest symptoms of the diseases of the dental structures and particularly the signs of susceptibility and immunity to dental caries. To do so one must be acquainted with the normally healthy mouth.

A small percentage of persons have continuously healthy mouths over long periods of time, some throughout their lives. They do not bother to brush their teeth for the reason that artificial cleaning is unnecessary. These persons seem to masticate their food with much vigor and whatever of food debris may remain in their mouths after a meal disappears completely within a short time. If the mouth is examined an hour after a meal, it is as clean as that of the most particular and fastidious person, who is very meticulous in the care of the mouth. The teeth of such persons are caries free and the gingivæ and supporting structures present a tone of splendid health, which corresponds to the perfect gloss of all surfaces of their teeth. Such persons have great bite force and use it at each meal; the supporting structures are in perfect condition, presumably because of the stimulating effect on the circulation which results from the vigorous use of their teeth. Their saliva is different, it seems to be lacking in the particular element which contributes to the formation of what have been termed gelatinous plaques or films, which adhere to the teeth.

THE SEVENTH EDITION.

This seventh edition is a combined revision of G. V. Black's Operative Dentistry and his Special Dental Pathology. The first edition of the Operative Dentistry was published in 1908, and the sixth edition in 1924. The first edition of the Pathology was published about six months before his death in 1915, and the fourth edition in 1924.

In this present revision, there has appeared no good reason to deviate from the principles laid down by G. V. Black in the original editions of these books. This work seems to have been so thorough as to stand the test of time.

NOMENCLATURE.

The nomenclature used in this work, in its main features, was developed by G. V. Black in a report * made by him before the World's Columbian Dental Congress in Chicago in 1893. Few changes have been made since the initial publication of the first editions of the Operative Dentistry and Special Dental Pathology. The following statements are quoted from the original introductory chapters:

Dentistry has its own nomenclature which has become distinct from the nomenclature of comparative dental anatomy. The nomenclature of dental anatomy from the standpoint of dentistry belongs distinctively to dentistry, and should in no case be confounded with the nomenclature of comparative dental anatomy, nor the one used in the place of the other, nor should any effort be made to harmonize them. When the human teeth are under consideration from the comparative anatomy standpoint, the nomenclature of comparative anatomy, which is suited to the description of the teeth of animals in general, should be used. In that nomenclature we do not speak of buccal and lingual surfaces of teeth but of inner and outer surfaces, the bicuspid in dental nomenclature become premolars in comparative dental anatomy. While there are points of coincidence in these nomenclatures, there are wide differences that could not be reconciled without positive injury to both.

The nomenclature of cavities and instruments "is simple, systematic and effective in teaching, in pointing out definitely the instruments and the manner or use of each. Every detail of cavity form is brought under a system of nomenclature comprised of a very few efficient rules, which are satisfactory for teaching purposes and for general use by practitioners."

"A nomenclature sufficient for a satisfactory description and clear understanding of the various parts of the gingivæ and peridental membrane and their functions has been developed. Particular attention is given to the various groups of fibers and their functions in maintaining the teeth in position under normal conditions and in the movements which result from the cutting off of certain groups of fibers by disease. The names of the diseases of the soft tissues indicate both the cause and the tissue principally involved. Such a nomenclature is essential to a proper understanding. Inflammations involving the gingivæ only are definitely separated from those involving the peridental membrane, as a basis for rational preventive treatment of diseases of the peridental membrane, because gingivitis is a necessary antecedent of these diseases."

* Report of Committee on Dental Nomenclature. Transactions of the World's Columbian Dental Congress, 1893, Volume II, page 835.

Only a few changes have been made in the nomenclature used in this revision, also a number of minor changes in the wording of definitions:

The term *cemental line* has been substituted for *gingival line* to obviate confusion from discussions as to the possible attachment of the gingival epithelium to the enamel.

Restoration is used to indicate all types of restorative operations—fillings, gold inlays, porcelain inlays, etc., etc.

Condenser is used instead of plugger.

Prepared Cavity is used to designate a cavity which is ready to receive a restoration, to distinguish it from a *cavity of decay*.

Resistance and *Retention* form are combined as an order of procedure in cavity preparation, because both are obtained with the same instruments at the same time. The distinction between resistance and retention form is not changed.

Starting point is substituted for *convenience point*, and the starting point is listed under retention form. This change was made to avoid confusion of the terms *convenience point* and *convenience form*.

Extension for Prevention is applied to the extensions of Class 1 cavities along fissures, as such extensions are made to prevent recurrence of decay.

The term *temporary teeth* is used in preference to *deciduous teeth*, because of its seemingly better relation to the term *permanent teeth*.

The term *successional teeth* is used in preference to *succedaneous teeth*.

The nomenclature of cutting instruments has been simplified by combining the *chisels* and *hoes* into one set of *chisels*, also by combining the *enamel hatchets*, *ordinary hatchets* and *special hatchets* into one set of *hatchets*.

ARRANGEMENT IN FOUR VOLUMES: CHAPTERS BY OTHER AUTHORS.

Attention is called to a number of chapters in this work, written by persons of special training and extensive experience in their respective fields. Each will be mentioned in the summary of the contents of the four volumes which follows.

VOLUME ONE.

This volume is devoted primarily to the pathology of dental caries, and other diseases of the hard tissues of the teeth. There are, however, several preliminary chapters of a general nature which are related to the contents of all four volumes.

ORAL DIAGNOSIS is the title of the first chapter, and there are presented the diagnostic data for the mouth lesions of more than one hundred and fifty diseases and abnormalities which are more or less frequently manifested in the oral cavity. This appears to be the first time that the oral expressions of systemic and skin diseases, and the symptoms of the diseases of the soft tissues of the mouth have been presented alongside the diagnostic signs of the strictly dental lesions. Some very interesting relationships are revealed, as for example, the inflammations of the gingivæ in the diseases of the blood forming organs. This chapter was written with the hope that it would be an incentive to the general dental practitioner to take full advantage of the frequent recalls of patients by making more critical examinations of the entire oral cavity. He should do so with the expectation that he will occasionally discover serious mouth lesions, such as cancer, certain of the diseases of the blood forming organs, or other conditions, previous to the time when the patient would be inclined to consult his physician. Specific directions are given for the making and recording of thorough examinations, and there are references to more than five hundred illustrations in other text books, devoted to diseases and abnormalities which are outside the strictly dental field.

HEALTH, NUTRITION AND HYGIENE is the title of a chapter by Clara Davis, M. D., pediatrician, who has devoted twenty-five years to the study of nutritional problems of children in the Mt. Sinai Hospital in Cleveland, and the Children's Memorial Hospital in Chicago. Her studies include a self selection diet experiment for which thirty-two thousand meals for ten children were very exactly tabulated and analyzed. Dr. Davis presents, in a most comprehensive way, the interrelation between the diseases of childhood and malnutrition and discusses the related problems of health and hygiene in connection with nutrition in such a manner as to make her presentation of great value to the dentist.

DEVELOPMENT OF THE TEETH, THE OCCLUSION, ETC. A new chapter has been prepared on the development of the teeth, the occlusion and related matters, which is limited to the consideration of those questions which have a definite bearing on the pathology and treatment of the strictly dental diseases.

THE SALIVA. There is a short chapter on the saliva by Earl A. Zaus, M.D., Professor of Physiology, and Harold L. Hansen, Ph.D., Professor of Chemistry, Northwestern University Dental School. This is in part a study, such as may be made by the dentist himself in noting certain conditions which may be related to susceptibility to caries. In the main, it is a summary of chemical and physiological studies, with which it seems desirable that the dentist should be familiar in view of the many investigations now in progress and their possible relation to susceptibility to dental caries, deposits of calculus, etc.

DYSTROPHIES. The developmental injuries of the hard tissues of the teeth, such as mottled enamel, hypoplasia, etc., are confined to failure in development of parts of the enamel and dentin because of general systemic conditions which interfere with nutrition at a time when some particular part of the tooth is being formed, or is growing, and the injury is confined to that part. Other tissues have the power of repair of such injuries later. Since the enamel and dentin do not have this power of self-repair, such injuries in them are permanent. There is a similar failure of self-repair in these tissues when injured by accident or disease after they have been formed, or have grown and completed their development, such as accidental breakage of parts or injuries by erosion or by caries. As these tissues are not subject to inflammation, nor to physiological or pathological changes in the same sense in which these occur in soft tissues and the bones, and as they are amenable to treatment only by artificial repair, it seems especially fitting and desirable that these be considered in a group to themselves.

Those conditions that occur in the form of malformations or misbuildings, such as supernumerary teeth, odontomes, malformed teeth, etc., belong to a different class and require totally different treatment.

MOTTLED ENAMEL is the title of a special chapter, with sixty illustrations, by Frederick S. McKay, D.D.S., who has tenaciously studied this peculiar endemic deformity for nearly thirty years and is at last able to write the practically complete story of the etiology, pathology and treatment. Dr. McKay's investigations have also developed the fact that this condition, although endemic, is affecting a larger number of teeth than the better known dystrophy to which the term hypoplasia is applied. Dr. McKay's chapter will be of special interest to dentists who practice in geographic areas in which the condition prevails. In all such areas, it becomes the duty of the members of the dental profession to insist upon a change or modification of the water supply to prevent the occurrence of this deformity.

HYPOPLASIA, or atrophy, is of such frequent occurrence, and the injury to the teeth is often so severe, that it must continue to be a source of concern, especially in the management of the first permanent molars and the incisors. The chapter by Dr. Davis emphasizes the nutritional damage which occurs in illness during early childhood and the reduction of cases of this type can result only from the better medical and nutritional care of children who suffer from diseases and malnutrition during the first five years of life.

EROSION is receiving more attention because the number of cases appears to be increasing, yet no new information of conse-

quence has been revealed in recent years. It still remains an enigma in the field of dental pathology.

ABRASION. Clinical studies of abrasion point to the need of earlier treatment than has been customary, to limit the lateral and protrusive movements of the mandible as the best, and in fact, the only means of reasonably controlling this condition.

DENTAL CARIES and its treatment must ever continue to be the most important subject in conservative dentistry. The locations and beginnings of decay, the physical injury to the enamel and the penetration of the dentin are well understood and the mechanical treatment is well systematized. Further improvement in treatment can not be achieved without better understanding of those conditions which control susceptibility and immunity. Continued studies of nutrition hold many possibilities and the investigations of recent years point toward the finding of an index of susceptibility in the environment of the teeth.

VOLUME TWO.

This volume is devoted to the materials and appliances used in operative dentistry, the nomenclature of cavities and instruments and the technical procedures in making restorations. For the teacher and student it is a presentation of the preclinical training, which should be given for the most part in laboratories of applied physics and operative technics.

MATERIALS USED IN OPERATIVE DENTISTRY. This special chapter was prepared by Eugene W. Skinner, Ph.D., Professor of Physics, Northwestern University Dental School. The physical properties of the materials and the principles of metallography and manipulation are presented for dental amalgams, gold foil, inlay casting gold alloys, dental porcelain, dental cements and gutta-percha. The student thus has the opportunity to become acquainted with the properties of these materials before he receives training in their manipulation. Dr. Skinner has recently published a separate text on the "Materials Used in Dentistry."

TECHNICAL PROCEDURES IN MAKING RESTORATIONS IN THE TEETH. This presentation is designed to follow courses in oral anatomy, the materials used in operative dentistry, and the pathology of dental caries, thus completing the preclinical training in this field. System and exactness in every detail of procedure is here emphasized. A new set of cutting instruments is proposed in which the classification is simplified and a few new forms are included.

The technical procedures in cavity preparation are preceded by a brief histological study of the structure of the teeth and there is interwoven only as much of the pathology of caries as seems to require repetition in connection with the laboratory instruction

in cavity preparation. It is recommended that the main features of the pathology of caries should be presented in the freshman year to impress the fact that caries is definitely a disease and give the student a reasonable understanding of the physical injury to the enamel and dentin.

It is intrinsically wrong to consider the treatment simply from the mechanical standpoint; it is wrong in that it tends to produce in the minds of students the idea that the making of restorations in the teeth is a purely mechanical pursuit. This is far from the proper conception of the facts. The closest use of knowledge of the pathology of dental caries and of the local conditions of its occurrence, should be made in every case, in order that the greatest benefit may be derived from these operations.

In presenting the technical procedures, the details of the adaptation of instruments in cavity preparation have been brought into close systematization through notes of practical work at the chair representing actual operative experience. The careful classification of cavities and of instrument forms adapted to each make it possible to teach cavity preparation in a way that it is easily learned; cavities may be more easily prepared, the time consumed is shortened, the operation is more definite in its results.

Operative dentistry, particularly when closely pursued for years together, is extremely taxing upon the nervous system of the operator, and many men break themselves down purely through assuming positions at the chair that are unnecessarily fatiguing. This arises from assuming wrong positions in the beginning and the failure to obtain that relief which is clearly and easily possible by change and the rest that change brings, without ceasing or slowing the work at the chair. Finger positions and positions at the chair are inseparable; one can operate better, faster and with less fatigue if these matters are carefully studied.

The chapter on the use of the rubber dam and other means of keeping the operating field dry has been largely rewritten to impress the needs of a dry field in connection with present day procedures. The tendency on the part of some practitioners to use less effective means than the rubber dam is resulting in the failure of many otherwise excellent operations.

VOLUME THREE.

THE CLINICAL TREATMENT OF DENTAL CARIES is presented in this volume and the effort has been made to bring together and coordinate as far as seems practicable all methods and procedures which may be employed. It is therefore designed as a *clinical guide, both in teaching and in practice*, and patient management becomes a prominent feature, made possible by the presentation of the material in the form of the clinical reports of cases in practice.

THE CARE OF THE TEMPORARY TEETH has been given the prominence which this service deserves. As one endeavors to analyze the dental diseases and their causes, he is inevitably carried back to the neglect of the temporary teeth and statistics are accumulating which will support the contention that the conservation of the permanent teeth depends to a very large extent on the most painstaking care of the temporary teeth.

A PROGRAM OF TREATMENT is presented which includes the education of patients and laymen generally in problems of dental health, the training of patients in mouth hygiene, the use of preventive and immunization measures by the dentist, and the reduction of pain in cavity preparation, all in proper relation to the making of restorations for individual patients "in the chair." This includes also the selection of the material to be used for the immediate restoration.

CLINICAL PRACTICE is divided into three periods: that of the temporary teeth, that of the permanent teeth during childhood and early adult life, and that of the adult. The differences in the problems of caries susceptibility and patient management are rather clearly drawn for each period and the relations of childhood care to adult conditions are stressed in a way that would be impossible in abstract discussions of technical procedures. An intimate dentist-patient relationship is established and maintained for a small group of patients over a long period of years.

The fact has been kept prominent that immunity to dental caries will become established in early adult life in the larger proportion of cases in which effective protection has been given by proper operations, by continuance of active mastication of food, and reasonable care as to cleanliness. This is so, as applied to the majority of persons who are very susceptible during the childhood period.

VOLUME FOUR.

This volume is devoted to the diseases of the gingivae and peridental tissues and the diseases of the dental pulp and periapical tissues. Diseases of the peridental membrane occur as the result of either a preceding gingivitis, which first involves the peridental membrane at the cemental line of the tooth; or the death of the dental pulp, which first involves the peridental membrane at the apex of the root. From other than these two points of beginning, disease of the peridental membrane does not occur, excepting as a result of some unusual traumatism. In both groups the investing tissues of the teeth are involved, and this makes it especially advantageous to present a careful study of the physical functions of these tissues.

It is also advantageous to correlate the pathology of these two groups, as they include practically all of those foci in the mouth

which endanger the general health. In the chronic suppurations of the peridental membrane beginning at the cemental line and in many cases of chronic periapical inflammation, the investing tissues are detached from the cementum, and in the treatment of both we are confronted with the same problem of the impossibility of repair, due to the peculiar characteristics of the cementum. When such detachment occurs, the pus-soaked cementum becomes practically a dead tissue, which can not be exfoliated, and therefore maintains the chronic focus indefinitely. The relationship of these chronic foci to serious secondary lesions demands the elimination of these foci, as well as the institution of more effective operating for their prevention in the future.

THE DISEASES OF THE GINGIVAE AND PERIDENTAL MEMBRANE, as well as the various causes, are separately considered, both as to pathology and treatment. It is of the utmost importance that these different conditions be recognized as a basis for correct diagnosis and proper treatment. The dentist who is able to make an exact and full diagnosis of the various diseases will have little difficulty in determining the best course to pursue in treatment.

Studies of deposits of salivary calculus have shown that these deposits and the damage to the tissues caused by them may be entirely prevented by proper brushing. This suggests the need for the special training of patients in the brushing of the particular regions where the deposits occur.

Deposits of serumal calculus are recognized as a cause of gingivitis when they occur on the enamel, but in cases in which pus pockets have been formed, it will be shown that these deposits occurring on the cementum are never a primary cause, but rather a result of the pocket formation.

Anyone who will make a careful analysis of several hundred cases of gingivitis in young adults will be convinced of the fact that the very large majority of cases, which progress to serious peridental infections, are due to local trauma. Some variation from the normally tight contact, particularly in the bicuspid and molar region will account for about half of all cases of peridental disease. In fact, it may be said that chronic suppurative pericementitis rarely occurs in mouths in which the dentist has seen to it that good contacts are maintained and the patient gives his mouth reasonable care in the matter of brushing.

The treatment of these cases, when properly diagnosed is usually simple, offering the key to prevention of this most destructive of dental diseases. Effective prevention of the diseases of the investing tissues must be brought about by greater perfection of those operative details which will promote the continued health of the gingivae.

DISEASES OF THE PERIDENTAL MEMBRANE, beginning at the gingival margins, are perhaps, of all the diseases of the dental

tissues, the least well understood. This is because of an insufficient knowledge by practitioners of the histology, physical functions and special physiological relations and dependence upon each other, of the tissues involved, and the failure to study the local causes leading to the establishment of the disease by any efficient system of records of cases in order to note their origin and progress.

There has been some confusion of ideas regarding the pathology of these diseases. This is largely because of the slowness of their progress. Any disease which progresses so slowly is especially difficult to study in its completeness. It is not like the study of, for instance, the acute form of alveolar abscess, which may begin and run its course in from two to six days, and one easily gathers the essential symptoms. In diseases of the periodontal membrane, beginning at the gingival margin, in which the rise and progress usually extend over a number of years, often without symptoms of which the patient will complain, the difficulties are greatly increased.

Studies of the investing tissues and of their reactions to chronic infection have led to the gradual development of practical methods of treatment which make it possible to entirely eradicate infection and conserve the teeth indefinitely without jeopardizing the patient's health. The technic of pocket elimination by gum resection has been so simplified that this operation should be performed as a routine procedure by the general practitioner.

Conditions in the mouth are such that it is impossible to maintain asepsis. This fact, coupled with the fact that detachments of the periodontal membrane from the cementum produce a constantly acting irritant, place these diseases in a class by themselves, entirely different from suppurations which occur elsewhere in the body. In treatment we should appreciate the exceptional powers of the mouth tissues in combating infections and should encourage them by maintaining the limit of cleanliness, rather than hinder them by the use of drugs which interfere with their activities.

THE DISEASES OF THE DENTAL PULP, beginning with hyperemia and continuing through the entire range of inflammations of the pulp and periapical tissues to and including acute alveolar abscess and necrosis, are presented in much detail, with their treatment.

In the consideration of the dental pulp, enough of the histology and physiological functions are given to enable the reader to gain the best understanding of the diseases of this tissue. It will be noted that the classification of these diseases is based upon the clinical manifestations, rather than upon microscopical examinations, which can not be satisfactorily applied in practice.

The chronic inflammations of the periapical tissues are given special consideration, for the reason that they present unusual complications in both diagnosis and treatment. It is the dentist's

duty to keep the mouths of his patients free from chronic infections without unnecessary sacrifice of teeth.

Emphasis is placed upon a program of patient management by which pulp involvements can be greatly reduced. The large number of persons who have been cared for according to this program up to the twenty-fifth year, without the loss of a pulp, is proof of its effectiveness. A group of fifty children in one orphanage in Chicago was cared for from the time of the eruption of the first permanent molars for a period of ten years with the loss of pulps from only three permanent teeth and the loss of four other teeth in which the pulps became exposed.*

The technic of all procedures in pulp treatment, including measures for the best asepsis, are given in great detail.

RADIOGRAPHY has contributed to much more accurate diagnoses of conditions within the maxillary bones than was possible previous to its use. The employment of the radiograph in the examination of cases of chronic suppurative pericementitis shows clearly the progressive absorption of the alveolar process subsequent to detachments of the peridental membrane from the cementum. The showing of cavities within the bone about the ends of the roots brings home the importance and absolute necessity for more careful technic and greater thoroughness in the handling of root canals. This should also impress the need for more accurate diagnoses of pulp conditions and much greater consideration for the continued vitality of the pulp in all restorative procedures.

MOUTH INFECTION IN RELATION TO SYSTEMIC DISEASE. This volume contains a special chapter on mouth infection in relation to systemic disease by Edward H. Hatton, M.D., Professor of Pathology and Bacteriology, Northwestern University Dental School. In this, Dr. Hatton has reviewed briefly the development of knowledge of the relations of foci of infection to secondary manifestations, including the part which oral foci appear to play. The structural changes which occur in the tissues, from their normal arrangement to that of the diseased condition, are described and illustrated. The further discussion endeavors to assist the practitioner in answering the question with which he is confronted almost daily—what teeth shall be extracted?

GLOSSARY.

The glossary, heretofore published in Volume I of the Operative Dentistry, has been expanded to include the terms used in connection with the diseases and treatment of the pulp and peridental tissues, also the terms used in the special chapters included in this edition. It is published in Volume IV.

*Honoroff, H. A., Jnl. Am. Den. Assn., Vol. 21, 1934, p. 37.

ILLUSTRATIONS.

The illustrations in this work consist of the large majority of those heretofore published in the Operative Dentistry and the Special Dental Pathology, to which have been added new photographic reproductions and original drawings by the present author to the number of about six hundred. There are a limited number of reproductions from other publications, for which due credit is entered in each instance.

In order to enable the student to more readily visualize the instrumentation in cavity preparation and restorative procedures, the positions of the various instruments are shown for each step in proper sequence for the cavities of each class in both the temporary and permanent teeth.

Particular attention is called to the references in the chapter on Oral Diagnosis, to more than five hundred illustrations in other text books, which treat of lesions outside the strictly dental field.

ORAL DIAGNOSIS

34 ILLUSTRATIONS: FIGURES 1-6E.

IN GENERAL it may be said that a greater variety of diseases involve the mouth than any other single part of the body, consequently diagnosis, as it affects the mouth, is a problem of much importance. In this chapter, the diagnostic data are presented for 152 diseases and abnormalities which find expression in the mouth and, in addition to the large number of illustrations in the four volumes of this work, references are given to more than five hundred illustrations in other books dealing with diseases of the oral cavity.

This chapter is included in this work on operative dentistry, written for the general dental practitioner, for three reasons; first, to broaden his knowledge of the scope and possibilities of dental service; second, that he may have fuller appreciation of the problems which confront the physician in the treatment of disease in other regions of the body and be prepared to give better cooperation; third, to assist him in the diagnosis of the more common diseases of the mouth, to the end that he may occasionally discover a serious lesion sufficiently early to render a splendid service by referring the patient to a physician for the prompt employment of effective treatment. He may thus assist in cutting short a disease which might otherwise impair the health and possibly threaten the life of the patient. Between three and four per cent of deaths from cancer occur as a result of mouth lesions. Many of these patients can be cured and the dentist often has the opportunity to discover cancer and other diseases of the mouth early, by reason of the fact that he sees his patient more frequently than does the physician.

DIAGNOSIS is defined as the determination of the nature of disease. It is the act or art of identifying a condition which may be any departure from the normal. It is the focal point on which all studies of the basic sciences and those of pathology and related conditions center, and from which all considerations of treatment radiate. See Figure 1. In making a diagnosis, one must apply in reverse order the knowledge gained in the study of the basic sciences.

KNOWLEDGE OF BASIC SCIENCES NECESSARY TO AN UNDERSTANDING OF DISEASE. The science of biology, through its departments

of anatomy, histology, physiology and chemistry, provides knowledge of the various cells and their combinations and correlations in the many bodily structures in the normal development, growth and activity of the body. Pathology, with the assistance of bacteriology and chemistry, through its divisions of etiology, morbid anatomy and symptomatology, supplies information as to the changed conditions produced by disease, and is necessarily based upon an understanding of the normal. In the division of nosology the various diseases are classified.

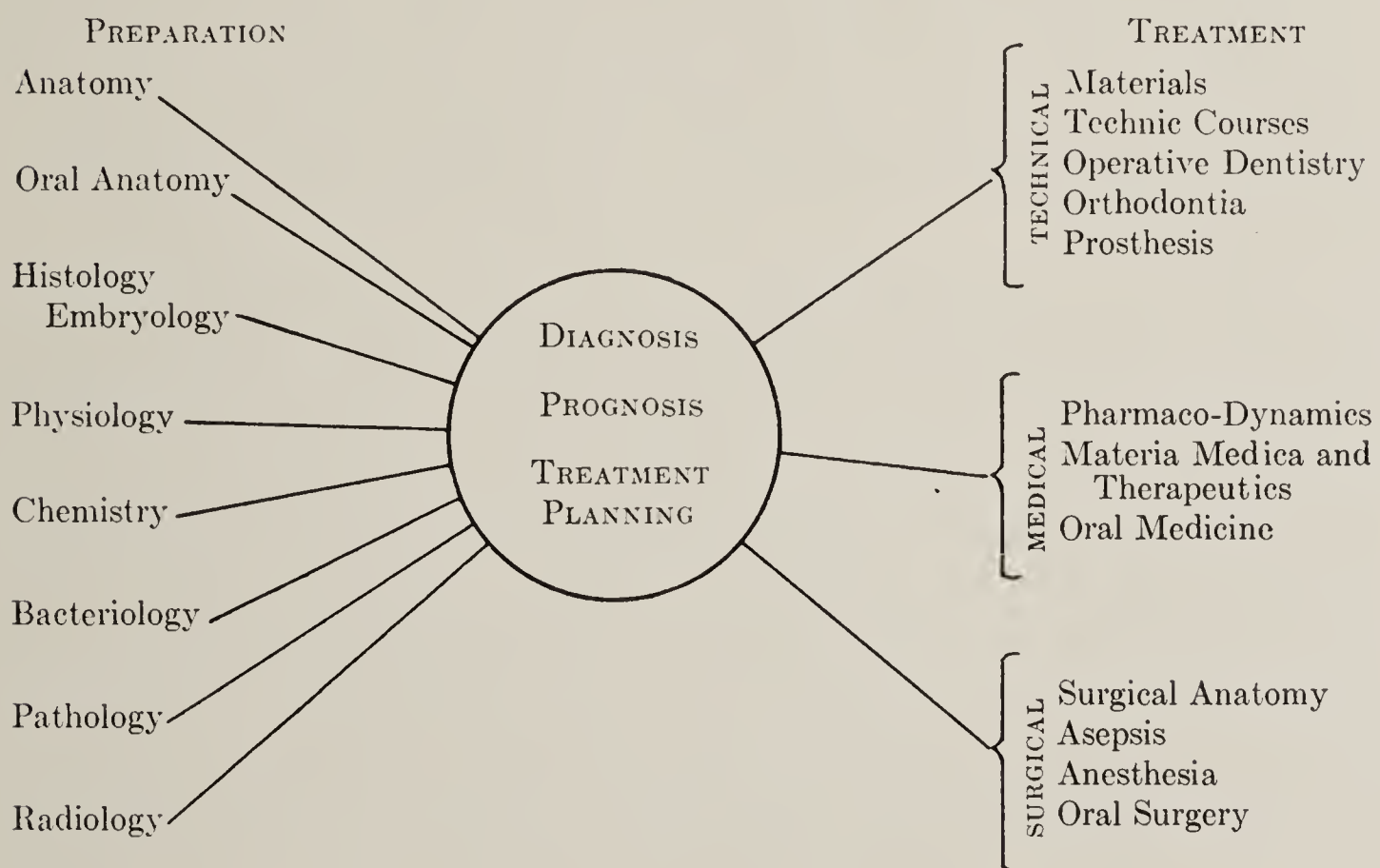


FIG. 1. Diagram illustrating the importance of studies in the basic sciences and in pathology and bacteriology as a necessary preparation for making diagnoses, prognoses and for the planning of treatment. It also illustrates the relation of other courses of study to clinical treatment; technical, medicinal and surgical.

THE DIAGNOSIS. In practice, one is confronted with a diseased condition, and must begin with the signs and symptoms to analyze the changes and arrive at a diagnosis—a classification of the particular disease—which results from an analysis and correlation of all of the variations from the normal. The diagnosis may eventually be based on any one or several of a wide variety of findings, such as changes in cells, a chemical analysis of a body tissue or fluid, a specific microorganism, a change of facial expression, the posture or gait of the patient, the mental or nervous reactions, the shadows of a radiograph, the detection of abnormal sounds within the chest, the temperature, the patient's description of painful sensations, the discovery of areas of tenderness or swellings; on any combination of the varied evidences of disease that may be found as a result of orderly procedure in diagnosis.

THE PROGNOSIS is a forecast as to the probable course, duration and outcome of a particular attack of a disease. This is based upon the patient's general physical condition, an estimate of the gravity of the signs and symptoms, also the apparent and possible complications in the case under consideration, and the treatment to be employed.

TREATMENT PLANNING. The planning of treatment is based on the diagnosis and prognosis. This is important in dentistry as in other fields of practice. In determining upon a program of treatment, several methods may be considered for a particular case and the value and probable result for each must be weighed against the others. In many cases, the sequence of treatment is of importance in securing the most satisfactory results, with the least discomfort and inconvenience to the patient while the service is in progress.

TRAINING FOR TREATMENT. In dentistry, the qualifications of the practitioner for the treatment of diseases of the teeth and other mouth tissues is developed along three rather distinct lines, all of which are coordinated with the basic science studies already mentioned. First, technical treatment, which begins with a study of materials, followed by technical laboratory courses and then clinical treatment in the fields of operative dentistry, orthodontia and prosthesis; second, medicinal treatment, based on studies in pharmacodynamics, materia medica and therapeutics, followed by treatment in the field of oral medicine; third, surgical treatment, based on studies of surgical anatomy, asepsis and anesthesia, followed by treatment in the field of oral surgery. Preventive measures are applied in greater or less degree to all conditions and as a part of practically every method of treatment. Technical services in the fields of operative dentistry and orthodontia have been developed with consideration of the preventive phases of each procedure, and prosthetic appliances are so designed that they give the best possible protection to the remaining teeth and the soft tissues.

METHODS USED IN MAKING EXAMINATIONS.

As a rule, in the examination of persons who apply to a physician for treatment, the patient is first requested to tell of his condition in his own words. His story will usually suffice to enable the physician to ask questions which will elicit a complete record of the patient's observations. The personal medical history of the patient, and more or less of the family history will also be recorded in all cases in which these may have a bearing on the immediate illness.

A general physical examination will then be made. This will begin with a record of the sex, age, height, weight, general appear-

ance, etc., followed by a more critical inquiry into the symptoms and signs which may be elicited, followed by radiographic and laboratory examinations, also various tests which may seem desirable in the particular case. In medical literature, the word symptoms is commonly used to include both the symptoms and signs of a disease.

SYMPTOMS. These are the phenomena observed and reported by the patient, such as pain, tenderness, dizziness, nausea, etc. It will often require careful questioning to determine the exact nature of pain. Special attention will be given to this symptom in considering the diagnosis of pain by the dentist.

SIGNS are objective evidences of disease, as follows:

Inspection. Visual examination.

Palpation. The use of the fingers applied to the surface of the body with light pressure, to determine the consistence of the tissues beneath.

Percussion. The striking of the part with sharp, short blows to determine the condition of the parts below by the sound obtained.

Auscultation. Listening to sounds within the body; used chiefly to ascertain the condition of the lungs, heart, pleura, and other organs; also conditions during pregnancy.

Pulse; rate and quality. Usually determined by applying the fingers to the radial artery at the wrist.

Temperature. Normal, 98.6°F, with which variations above or below are compared.

Respiration. In health, the rate varies from 13 to 25 per minute, the average being about 19. Notation of variations in rate and quality.

Blood pressure. The pressure of the blood on the walls of the arteries, which depends on the energy of the heart action, the elasticity of the vessel walls, the resistance of the capillaries, and the volume and viscosity of the blood. The systolic pressure is the maximum, the diastolic is the minimum pressure. These are determined by measuring the force required to compress the brachial artery until the pulsation is not registered.

Basal metabolism. The minimal heat produced by an individual, which represents the energy expended in maintaining the basal metabolic changes in the body. It is made with a calorimeter, and is called the basal metabolic rate.

RADIOGRAPHIC EXAMINATIONS. The radiograph gives much diagnostic information in the study of conditions within the body in which there have occurred sufficient changes in structure to produce abnormal shadows on the film. It is also used to locate foreign bodies which are opaque to the x-ray.

LABORATORY EXAMINATIONS. Various types of laboratory examinations are made to assist in arriving at a diagnosis. These include bacteriologic studies of fluids, smears and tissue; microscopic studies of tissues removed in operations, particularly when malignancy is suspected; the examination of urine for signs of diabetes and nephritis; blood examinations, such as hemaglobin tests and red cell counts in anemia, white cell counts in cases of infection, differential counts in leukemia, and chemical tests for calcium and phosphorus, etc.

THE DIAGNOSIS results from a critical study of symptoms and signs associated with the particular condition, together with a knowledge of the causal factors, etc. Several methods are commonly employed in making a diagnosis. The choice will depend upon conditions presenting. Oftentimes a combination of two or several methods will be used.

Clinical diagnosis, based on the symptoms and signs shown during life, irrespective of the morbid changes producing them.

Differential diagnosis, which consists of distinguishing between two allied diseases by contrasting their symptoms and signs.

Diagnosis by exclusion, which consists of the elimination of other diseases by the fact that some, at least, of the symptoms of each of the other diseases under consideration are not present.

Laboratory diagnosis, which results from the examination of various body tissues, fluids and secretions in the laboratory.

NEED FOR BETTER DIAGNOSTIC METHODS IN DENTISTRY. The adoption of methods which make for better understanding of the normal functions, as well as the pathologic changes and general systemic conditions in connection with diseases of the mouth, particularly in the early stages, are of much importance to the general practitioner of dentistry. They have the same practical bearing on treatment, both medicinal and technical, as do similar data in the other fields of health service. For many conditions in the dental field, as in every other specialty, elaborate programs are not necessary to arrive at a diagnosis; for others, various factors should have consideration and it is necessary to follow orderly methods. Accurate diagnosis requires a knowledge of normal structure and function, and of pathologic changes, also keen observation and the experience and ability to make proper deductions.

List of Diseases of the Mouth

The various diseases which affect the mouth, and other conditions which require consideration in diagnosis, which are included in this chapter, are arranged in several groups. For each disease the more important symptoms are given, also the differential diagnosis in cases in which that seemed desirable. Immediately following the diagnostic data of each condition, references are given to illustrations in the several volumes of this work for those diseases included in operative dentistry, and to other texts for conditions which are assigned to other fields of dental practice, or are treated by the physician or surgeon.

Particular attention is called to the fact that the strictly dental conditions, which are regularly treated by the general dental practitioner, are presented separately from all others, as are the suggestions for recording the examination and the planning of treatment. Therefore, the presentation of diagnostic data for other diseases and abnormalities leads to no confusion; the diseases of any group may be considered without regard for the others, or they may be combined as a single study of diseases which involve the mouth.

The following is a list of the diseases included in this chapter. No effort has been made to compile a complete list of diseases that affect the mouth, but to select those which should be of most interest to the dentist. For each group page references are given for the diagnostic data and for the directions for recording.

DISEASES OF THE TEETH AND SUPPORTING STRUCTURES.

THE OCCLUSION. Diagnosis, p. 25; Recording p. 68.

Classification of malocclusion.

THE DENTITION. Diagnosis, p. 27; Recording, p. 69.

Congenitally absent teeth.

Missing teeth.

Unerupted teeth.

Impacted teeth.

Temporary teeth retained.

Supernumerary teeth.

HARD TISSUES OF THE TEETH. Diagnosis, pp. 31, 37; Recording, pp. 70, 73.

Dental caries.

Hypoplasia.

Enamel whorl.

Corrugated teeth.

White spots in enamel.

White enamel.

Mottled enamel.

Erosion.

Abrasion.

GINGIVAE AND PERIDENTAL TISSUES. Diagnosis, p. 40; Recording, p. 71.

General gingivitis.

Salivary calculus—gingivitis.

Serumal calculus—gingivitis.

Local trauma—gingivitis.

Excessive occlusal stress—pericementitis.

Chronic suppurative pericementitis.

Lateral pericemental abscess.

DENTAL PULP AND PERIAPICAL TISSUES. Diagnosis, p. 46; Recording, p. 72.

Hyperemia.

Acute pulpitis.

Chronic pulpitis.

Dead pulp.

Chronic hyperplastic pulpitis.

Calcifications growing free.

Calcifications attached to walls.

Traumatic apical pericementitis.

Acute apical pericementitis.

Chronic apical pericementitis. Granuloma.

Chronic apical (alveolar) abscess.

Periapical cysts. Radicular cyst.

Acute periapical (alveolar) abscess.

PAIN ASSOCIATED WITH ORAL LESIONS.

Diagnosis, p. 81; Recording, p. 77.

Trigeminal neuralgia.
Glossopharyngeal neuralgia.
Atypical neuralgia.

Hysteria.
Phobias.

FOCI OF INFECTION IN THE MOUTH.

Diagnosis, p. 87; Recording, p. 77.

Chronic pericementitis.
Chronic apical infection.

Other chronic inflammations.
Acute periapical abscess.

SURGICAL DISORDERS OF THE MOUTH, JAWS AND SALIVARY GLANDS.

ABNORMALITIES. Diagnosis, p. 89; Recording, p. 77.

Cleft palate and hare-lip.
Torus palatinus.
Abnormal superior labial frenum.
Abnormal lingual frenum. Tongue-tie.

Unerrupted teeth. See p. 27.
Impacted teeth. See p. 27.
Supernumerary teeth. See p. 27.
Retrusion of the mandible.

HYPERTROPHIES. Diagnosis, p. 90; Recording, p. 77.

Prognathism.
Hypertrophic gingivitis.

Macrocheilia. Lymphangiectasis.
Acquired blood-vessel varices.

DENTAL INFECTIONS AND SEQUELLAE. Diagnosis, p. 90; Recording, p. 77.

Traumatic apical pericementitis. See p. 48.
Acute apical pericementitis. See p. 48.
Chronic apical pericementitis. Granuloma.
See p. 48.
Chronic apical (alveolar) abscess. See p. 48.
Periapical cyst. Radicular cyst. See p. 49.
Acute apical (alveolar) abscess. See p. 50.
Condensing osteitis. Osteosclerosis.
Periostitis.
Acute osteitis.

Chronic osteitis.
Osteomyelitis.
Necrosis.
Phosphorus necrosis.
Arsenical necrosis.
Acute maxillary sinusitis.
Chronic maxillary sinusitis.
Acute cellulitis. Ludwig's angina.
Actinomycosis.

SALIVARY GLANDS AND DUCTS. Diagnosis, p. 94; Recording, p. 77.

Salivary fistula.
Salivary calculi.

Ptyalism.
Atyalism. Xerostoma.

TEMPERO-MANDIBULAR JOINT. Diagnosis, p. 94; Recording, p. 77.

Dislocation of mandible.
Subluxation of mandible.
Trismus.
Arthritis.

False ankylosis.
True ankylosis.
Joint structure injuries and impaired hearing.

FRACTURES OF THE MANDIBLE AND MAXILLAE.

Diagnosis, p. 96; Recording, p. 77.

Fractures of the mandible.
Fractures of the maxillae.

Pathological fractures.

FACIAL DISORDERS. Diagnosis, p. 98; Recording, p. 77.

Facial paralysis. Bell's palsy.

Facial tic.

TUMORS. NEOPLASMS. CYSTS. ODONTOMES. Diagnosis, p. 99; Recording, p. 77.

Naevi. Moles. Birthmarks.
Epithelial tissue tumors.
Papillomata.
Adenomata.
Fordyce's disease.
Carcinomata.
Connective tissue tumors.
Lipomata.

Chondromata.
Myxomata.
Osteomata.
Myomata.
Epulis granulomatosa.
Fibromata.
Benign giant cell epulis.
Central benign giant cell tumor.

Connective tissue tumors (Con't).

Keloids.
 Sarcomata.
 Endotheliomata.
 Angiomata.
 Lympho-sarcomata.
 Hodgkin's disease.
 Tumors of salivary glands.

Cysts.

Mucous cysts.
 Ranula
 Radicular cyst. Root cyst. See p. 49.
 Follicular cysts.
 Odontomata.
 Adamantanoma. Multilocular cyst.

DISEASES OF THE SOFT TISSUES OF THE MOUTH.

Diagnosis, p. 108; Recording, p. 77.

Acute ulcerous gingivitis. Vincent's stomatitis.	Bismuth stomatitis.
Vincent's angina.	Lead stomatitis.
Agranulocytic angina.	X-Ray burns.
Thrush.	Angioma.
Aphthous stomatitis. Cancer sore.	Fordyce's disease. See p. 100.
Decubital or pressure ulcers, neurotrophic ulcers.	Geographic tongue.
Herpes simplex. Herpes labialis. Cold sore. Fever sore.	Black tongue.
Leukoplakia. Smoker's patches.	Burning tongue.
Mercurial stomatitis.	Macroglossia. Lymphangioma.
	Perlèche cheilitis.
	Cheilitis exfoliativa.

SKIN DISEASES; MOUTH MANIFESTATIONS.

Diagnosis, p. 115; Recording, p. 77.

Allergy.	Herpes zoster. Shingles.
Urticaria. Hives. Nettle rash.	Erythema multiforme.
Lichen ruber planus.	Acne vulgaris.
Phemphigus vulgaris.	Lupus erythematosus.

SYSTEMIC DISEASES; MOUTH MANIFESTATIONS.

DISEASES OF BLOOD FORMING ORGANS. Diagnosis, p. 119; Recording, p. 77.

Pernicious anemia. Hunter's glossitis. Moeller's glossitis. Glazed tongue.	Scurvy.
Leukemia.	Purpura.
Agranulocytic angina.	True hemophilia.

BONE DISEASES. Diagnosis, p. 122; Recording, p. 77.

Rickets.	Acromegaly.
Osteitis fibrosa.	Osteitis deformans.
Arthritis deformans.	

ACUTE INFECTIOUS AND CONTAGIOUS DISEASES.

Diagnosis, p. 124; Recording, p. 77.

Anthrax.	Typhoid fever.
Small pox. Variola.	Measles.
Scarlet fever. Scarlatina.	Diphtheria.
Influenza.	Epidemic parotitis. Mumps.

DIABETES. Diagnosis, p. 126; Recording, p. 77.

TUBERCULOSIS. Diagnosis, p. 126; Recording, p. 77.

Lupus vulgaris.

SYPHILIS. Diagnosis, p. 127; Recording, p. 77.

Examinations of the Mouth

The mouth examination should be made for the purpose of recording the pathological conditions discovered, rather than to list the technical procedures to be performed, although the latter may be added.

The importance of very painstaking and thorough examinations of the mouth can not be overemphasized. The service of the dentist will increase in effectiveness toward prevention as he learns to recognize the beginnings of disease. This applies to general conditions manifested in the mouth quite as much as to diseases of the dental structures. In proportion as decays of the teeth are discovered early, will cases of pulp death and alveolar abscess be diminished; likewise, as inflammations of the gingivae are recognized and treated early, will serious disease of the supporting structures be prevented; and in turn, the patient's health will be protected.

The record of an examination is of almost as much importance as the examination itself, by reason of the fact that the habit of recording necessitates a clear mental picture of the condition to be recorded. This begets keener observation and develops the faculty of logical thought and proper deductions, as applied to the conditions presenting. The dentist who develops the habit of making accurate records will soon learn to see as much more in mouth examinations as does the trained microscopist as compared with the beginner. The record will often be of much value in the later treatment of cases, and the gradual accumulation of data should eventually be the basis of tabulated reports as a guide for future practice.

CLASSIFICATION OF PATIENTS FOR EXAMINATIONS. Persons who apply to the dentist may be placed in three groups: 1. Those who present themselves at more or less definite intervals for routine examinations, usually without any particular complaint. 2. Those who come because of some particular condition of pain or discomfort, which calls for a special examination and contraindicates a complete mouth examination at the time. Such an examination must often give particular consideration to the symptom of pain. 3. Those who are suffering from some condition that might be secondary to a mouth infection, in which case the examination will be concerned only with a search for foci within the mouth.

CONDITIONS INCLUDED IN THE ROUTINE MOUTH EXAMINATION. The diagnosis of those diseases and abnormalities usually included in the routine mouth examination will be presented in the following

pages; the method of recording these on the examination card will then be given, and thereafter the diagnosis of diseases which require special examinations. For the routine dental examination, the various conditions are grouped under the following headings:

The Occlusion.

The Dentition.

Lesions of the Hard Tissues of the Teeth.

Diseases of the Gingivae and Peridental Tissues.

Diseases of the Dental Pulp and Periapical Tissues.

The early discovery of dental caries and of the slightest inflammations of the gingivae is stressed, for the particular bearing that early discovery and prompt treatment have on satisfactory management of cases in conserving the teeth. The success of such a program is not dependent alone upon one's ability to make a proper diagnosis, but considerably more upon a full understanding of the sequence of pathological changes which occur in cases that do not receive prompt attention. As has been pointed out, this includes the eventual systemic effects of neglected dental lesions. The fuller realization of the inter-relation of dental caries and the diseases of the pulp and supporting structures of the teeth, which become so plainly manifest, as cause and effect, over periods of many years, should incline every dentist to make more comprehensive studies of mouth conditions.

The mouth examination must be elevated to a place of supreme importance in mouth care, and it is the duty of each dentist to inculcate in the minds of his patients the fact that painstaking examinations are the first logical step in prevention, as applied to both the mouth and the general health.

The Occlusion

The generally accepted plan of making a diagnosis of malocclusion of the teeth was originally proposed by Angle. It is based on the mesio-distal relation of the lower first molar to the upper on either side of the arch, and the condition of the incisor teeth—whether the upper incisors are irregular, protruding or retruding, and whether or not the lower incisors protrude. For the purpose of the general dental practitioner, the record of these conditions in any given case will be sufficient. The orthodontist will take impressions, prepare models and make a more critical study of the position of each tooth. He will also inquire into all related conditions and make a record of these.

Dr. Lischer suggested the terms mesioversion, distoversion, buccoversion, labioversion and linguoversion as applied to individual teeth that are out of their normal positions in any of the directions mentioned; also the terms supraversion (elongated),

intraversion (depressed), perversion (impacted), transversion (transpositions).

More recently he has added the term eugnathic, for the simpler anomalies of position of teeth in well developed jaws—cases which should usually be treated by the general dental practitioner; and dysgnathic, for complicated dento-facial deformities with malformed jaws, which should be referred to an orthodontist.

It should be a rule of the dental practitioner to note and record the occlusion of the temporary teeth at the first examination after all of these teeth have erupted. He should refer cases of serious malocclusion to an orthodontist with the thought that it may be possible to so treat the case that the permanent teeth will erupt in normal positions. In some cases, the general practitioner may make minor adjustments previous to the eruption of the first permanent molars, as suggested in Volume III, page 89.

The occlusion should be recorded at the first examination after the eruption of the first permanent molars, and at intervals thereafter until all of the permanent teeth are in position. When a new patient presents, no matter what the age, the conditions as to occlusion should be noted and recorded.

TABULATION OF THE SEVERAL TYPES OF MALOCCLUSION. In the column to the left, the first letter indicates the relation of the lower first molar to the upper on the right side; the second letter the first molar relation on the left side; the third letter the condition of the incisors, upper or lower. A dash is used to indicate the lower incisors, as M M - P.

	Lischer	Angle
N N N	Normal.	Normal
N N I	Neutroclusion, upper incisors irregular.	Class 1.
D D P	Bilateral distocclusion, labioversion upper incisors,	Class 2, Division 1.
D N P	Unilateral distocclusion, labioversion upper incisors,	Class 2, Division 1, Subdivision.
D D R	Bilateral distocclusion, linguoversion upper incisors,	Class 2, Division 2.
D N R	Unilateral distocclusion, linguoversion upper incisors,	Class 2, Division 2, Subdivision.
M M - P	Bilateral mesiocclusion, labioversion lower incisors.	Class 3, Division.
M N - P	Unilateral mesiocclusion, labioversion lower incisors.	Class 3, Subdivision.

It will be noted that the three letters not only indicate the classification by Angle or Lischer, but also, in cases in which the abnormality is unilateral, the side on which the relation is normal and the side that is in abnormal relation. The three letters are of

themselves sufficient, without mention of class, division, subdivision, or any special nomenclature. The record suggested here enables the dental practitioner to note the general conditions of the teeth as to occlusion in the simplest possible way. The orthodontist would make a more complete record.

EXCESSIVE OCCLUSAL STRESS. After the child reaches the age when orthodontic treatment would be inadvisable, and particularly during the adult period, the maintenance of equitable occlusal stress becomes an important factor in preventing movements of the teeth which may result in injury to the investing tissues. The conditions which result from excessive occlusal stress are considered in the discussion of the diseases of the gingivae and peridental tissues.

The Dentition

On the occasion of the first examination, the dentition of the patient should be fully recorded. If the first examination is made during the period of the temporary teeth, or at any time before all of the permanent teeth have erupted, the original record should be revised at each examination as changes occur. Radiographs are



FIG. 2A.

FIG. 2B.

FIGS. 2A and 2B. Examples of supernumerary teeth in the form of *cones*.

usually necessary to assure the exactness of the record as it refers to teeth congenitally absent, unerupted, impacted, or supernumeraries.

At each examination the card should show the dentition as it exists at the time. After the period of the temporary teeth, the following conditions should be noted: Teeth congenitally absent; missing teeth, consisting of those not due to erupt, temporary teeth recently shed and teeth that have been previously extracted; teeth unerupted at the time when they should be in their places in the arch; the delayed eruption of any such tooth, which may be noted by recording the age when it does erupt; impacted teeth; tempo-

rary teeth retained beyond the normal time of shedding; and supernumerary teeth.

CONGENITALLY ABSENT TEETH. The upper lateral incisors are frequently congenitally absent as are any or all of the third molars, and one or more of the bicuspid may be missing. A rather definite hereditary trend has been noted in many cases of absence of one or both upper lateral incisors. In cretinism, which is caused by atrophy or injury to the thyroid gland, the bones and jaws are slow in development and the teeth, both temporary and permanent, are late in erupting. The temporary teeth are usually retained beyond the normal shedding time, and a few or many of the permanent teeth may be missing.

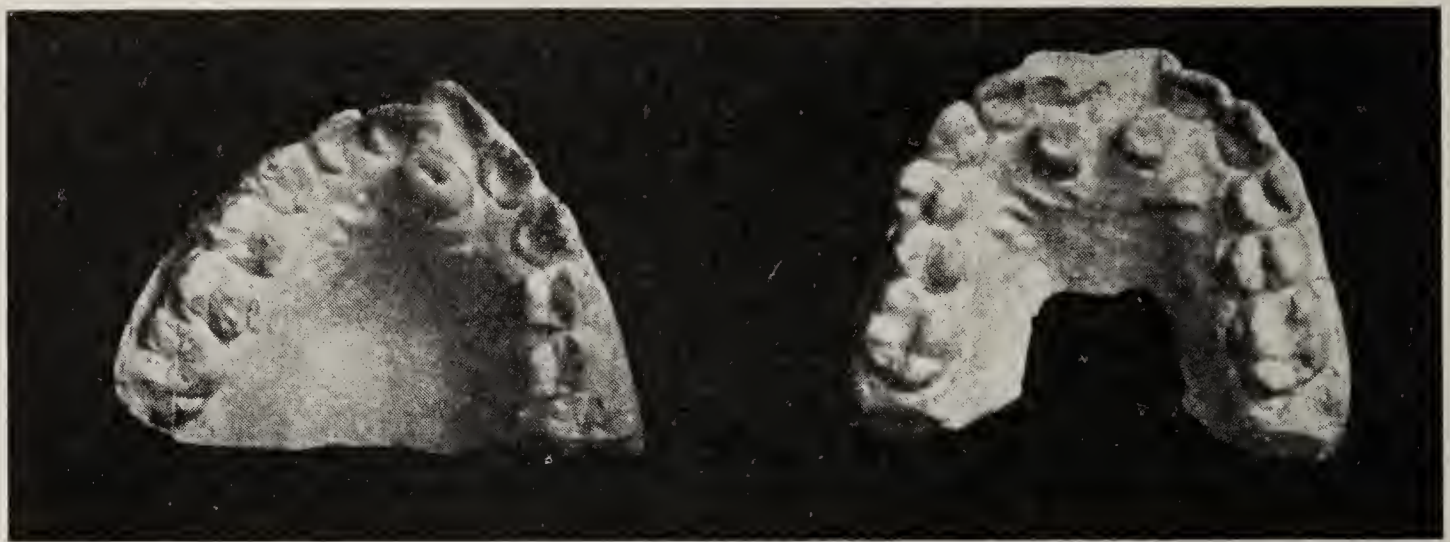


FIG. 2C.

FIG. 2D.

FIGS. 2C and 2D. Examples of supernumerary teeth in the form of *truncated cones*.

SUPERNUMERARY TEETH are formed by abnormal buds from the dental lamina. They may occur in almost any position, and may be of abnormal form or extra teeth of normal form. They may be classified* into six groups, as follows:

Cones. These correspond with primitive forms of teeth; they are usually small, of cone shape, and generally converge to a rather sharp point. They occur most frequently in the upper incisor region, and there may be one, two or several in the same arch. See illustrations, Figures 2A and 2B.

Truncated cone. These are similar in all respects to the conical form, except that the point of the cone is missing. The end may be slightly rounded or it may be slightly inverted, with a depressed center. See illustrations, Figures 2C and 2D.

Dichotome. This is a two-fold tooth. The term is applied to a plant that has two seeds in one pod, or a regular division into two

*See classification by G. V. Black, Dental Summary, Vol. 29, 1909, pp. 1 and 83.

branches. Here it is used to apply to a tooth that has two enamel germs, a supernumerary germ united with a normal enamel germ, there being a common pulp and root canal for both. These occur most frequently in the incisor region, upper or lower, and occasionally in the molar region. See illustrations, 2E and 2F, two views of the same specimen, and 2G.



FIG. 2E.

FIG. 2F.

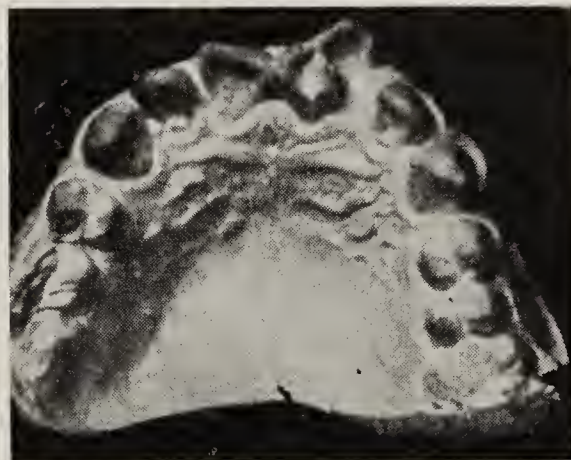


FIG. 2G.

FIGS. 2E and 2F. Two views of a *dichotome*, formed by the union of a large supernumerary tooth with a lower molar. The two teeth have a common pulp chamber.

FIG. 2G. Another example of a *dichotome* formed by the union of a supernumerary with an upper central incisor.



FIG. 2H.

FIG. 2J.

FIG. 2K.

FIG. 2M.

FIGS. 2H and 2J. Two views of *fused teeth*, a supernumerary united to a normal molar tooth by fusion of cementum.

FIGS. 2K and 2M. Another example of *fused teeth*; two conical supernumeraries united to the buccal side of a first molar.

Fused teeth. This name is applied to what might be termed double teeth, consisting of a supernumerary which is united with a normal tooth by a fusion of the cementum. Each tooth is complete in itself, having its own pulp chamber and root canals. These occur most frequently in the molar region. See illustrations, Figures 2H and 2J, two views of the same specimen, also 2K and 2M, two views of one specimen.

Two molars of the regular dentition, and occasionally all three, may be joined by a fusion of the cementum of their roots.

Gemma. The term *gemma* is applied to a supernumerary, the dentin pulp of which has united with the dentin pulp of an otherwise normal tooth. There may be several gemmae attached to a single tooth. There are practically always two distinct enamel caps, one supernumerary and one of a normal tooth, or two supernumeraries may be similarly joined. As a rule the two pulps have a common root canal and apical foramen. They are usually found in the molar region. See illustrations, Figures 2N, also 2P and 2Q, two views of the same specimen.



FIG. 2N.

FIG. 2P.

FIG. 2Q.

FIG. 2N. Example of a *gemma*; the roots of the supernumerary and the normal tooth are united and have a common pulp and root canal.

FIGS. 2P and 2Q. Two views of three conical *gemmae*, which are attached to the buccal roots of an upper molar. The root canals of all four teeth are united.



FIG. 2R.

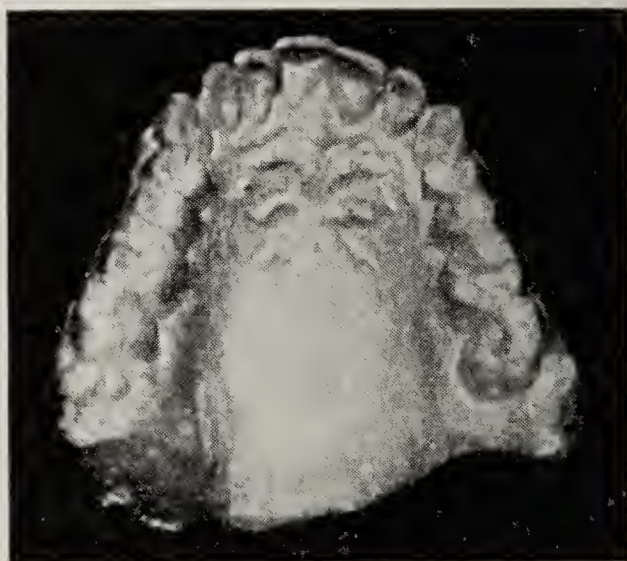


FIG. 2S.

FIGS. 2R and 2S. Examples of supernumerary teeth of *normal form*. In Figure 2R there are two supernumerary upper central incisors; in Figure 2S there is an extra upper lateral incisor on the right side.

Extra teeth of normal form. There occasionally occur supernumerary teeth of normal form; they may be incisors, cuspids, bicuspid or molars, although extra cuspids and bicuspid are less frequent than incisors or molars. An extra diminutive molar—a fourth molar, is of rather common occurrence. See illustrations, Figures 2R and 2S.

Lesions of the Hard Tissues of the Teeth

DENTAL CARIES. Decay, or caries, of the teeth is a chemical dissolution of the calcium salts, first of the enamel, then of the dentin, by lactic acid. It is primarily a disease of childhood and early adult life, although the degree of susceptibility may vary greatly for persons of all ages, and for the same person at different periods in life.

Decay always begins on the outer surface of the enamel and progresses inward through the enamel to the dentin, then continues inward toward the pulp, as well as laterally in the dentin along the dento-enamel junction, thus undermining the enamel and dissolving it from the inside outward. By this process a cavity is formed. Caries always begins in positions on the surfaces of the enamel of erupted teeth, where micro-organisms are so attached to the enamel that the lactic acid, which they form, is held in contact with the surface of the enamel and is not readily dissipated in the fluids of the mouth. The points of beginning are in pits and fissures, where the lobes of the enamel have failed to unite properly; in proximal surfaces, to the gingival of the contact point, and in the gingival third of labial, buccal and occasionally lingual surfaces.

A beginning area of caries is white when the tooth is dry. The whitened area, if occurring on a smooth surface, is etched and is somewhat rough to the point of a sharp exploring tine passed over it with slight pressure. With further progress, some of the enamel rods fall away and a cavity is formed. As the dentin is involved, it becomes soft in texture, and light yellow in color, often gradually changing to brown, and occasionally to black. Progress of decay in the dentin may or may not be accompanied by pain, which may be caused by the caries process itself, by thermal shock to the pulp from foods, drinks or cold air, or by pressure of food or any foreign substance within the cavity. The pain is usually greater as the decay approaches closer to the pulp, and greatest when the pulp is actually involved. Due to the fact that all of the teeth on each side of the mouth are innervated by branches of the second and third divisions of the fifth nerve, the patient may not be able to locate the pain in a particular tooth, or to tell whether it originates in the upper or lower jaw, unless the pain is due to pressure on the dentin in a fairly open cavity through the enamel.

DISCOVERY OF DENTAL CARIES.

PIT AND FISSURE DECAYS. The discovery of pit and fissure decays is usually a simple procedure. As the examination proceeds, the surfaces of the teeth in each bicuspid-molar region

should be dried with blasts of air, in order that the best view may be had. An exploring tine, with a small, sharp pointed end, should be used to test the positions of all pits, even though they appear not to be decayed. The point should be applied with some pressure and, if it enters the enamel a little, so that a very slight pull is required to remove it, the pit should be marked for a restoration, even though there is no sign of decay. There should be no question about the discovery of larger pit decays, as the surfaces of the teeth on which these decays occur are readily accessible. However, the size of the opening through the enamel, if small, may be no indication of the extent of the decay in the dentin. If the enamel immediately about the opening, particularly when dry, is greyish white, this generally is evidence of decay in the dentin, which has undermined the enamel. The lingual surface of the upper lateral incisors should always be examined, also the buccal and lingual surfaces of molars, as pits occasionally occur in these positions.

Illustrations: Volume I, Figures 154 to 158; 185 to 189; 194 to 196.

GINGIVAL THIRD DECAYS present little difficulty in diagnosis in cases in which some of the enamel rods have fallen away. However, when such a decay occurs in the teeth of a person who is recalled for examination at regular intervals, it should usually be discovered before any of the enamel rods have fallen away — before there is an actual cavity. Such an area appears as a white line close to the gum margin, or a white area, generally near the mesio-distal center of the buccal or labial surface, close to the gum. In looking for these areas, cotton rolls and the air syringe should be used to dry the surfaces, otherwise the decay may be overlooked.

Illustrations: Volume I, Figures 220 to 228.

PROXIMAL DECAYS IN BICUSPIDS AND MOLARS. Beginning decays in proximal surfaces are the most difficult of all to discover, particularly in bicuspids and molars with proximal surfaces which are somewhat flat, and in cases in which the crests of the gingivae have receded only a very little, so that the area of liability is very close to the point of contact. These decays must be discovered early, before the etching of the enamel has made much progress, if the preparation of a cavity and the placing of a restoration is to be avoided. It should be remembered that conditions conducive to the occurrence of proximal decays are likely to be much the same for most of the proximal surfaces of bicuspids and molars; therefore decays may occur in many of these surfaces at the same time or in rapid succession.

The surfaces of each group of teeth to be examined should be dried with a blast of air, blown through the interproximal spaces, to give the best possible view of both the enamel and gingivae.

The Use of Explorers. A pair of small right and left explorers with curved tines, sharply pointed, are very effective in detecting the slightest etchings of proximal surfaces. The instrument should be carried into the interproximal space with the handle at such an angle that the point of the instrument is directed occlusally, although at a slight angle to the mesio-distal axial plane of the teeth. This position permits the point of the instrument to be moved from buccal to lingual, and back again, while held very close to the contact point, there being room within the septal portion of the embrasure space to accommodate the curved portion of the tine. One instrument of the pair should be used to examine the mesial surface of each tooth from first bicuspid to the third molar, the approach being through the buccal embrasures. In many cases, the same explorer should then be placed in each lingual embrasure to examine the distal surfaces of the same teeth. The other explorer should be similarly used for the distal surfaces with the approach from the buccal, and the mesial surfaces with the approach from the lingual. By this plan each proximal surface will be examined twice.

If, in any interproximal space, there should be a question whether the close proximity of the two teeth prevented the explorer from being passed over the surface immediately to the gingival of the contact, or if there should be a sensation of the slightest roughness of the enamel, a Ferrier separator should be placed to move the teeth slightly apart, in order that there may be sufficient room to so manipulate the instrument that the condition of the enamel will be definitely determined.

The radiograph is extensively used for the discovery of proximal decays. The rays must be directed at exactly the correct angle through each interproximal space to prevent the slightest overlapping of the shadows of the enamel of the contact areas of the two teeth. When the etching of the enamel is sufficiently deep to cast a noticeable shadow on the film, it will usually be necessary to prepare a cavity and make a restoration. It will depend upon the expertness of the radiographer on the one hand and the skill of the dentist on the other, whether more small proximal decays will be found with radiographic films or with explorers. The radiograph is certainly of great value in the discovery of these cavities, but should not be relied upon to the exclusion of the instrumental examination.

Ammoniated Silver Nitrate may be used to disclose the slightest beginnings of decay. A plan of applying this solution for the immunization of decays of the enamel, the technic of which is identical with that for the discovery of caries, is presented in Vol. III, page 49.* It appears to be desirable that silver nitrate

*See also J. M. Prime, Dental Cosmos, Vol. 77, 1935, p. 1046. The illustrations on this and the following pages are from Dr. Prime's article, except Figures 3E, 3F, and 3G, which were prepared by Dr. W. G. Skillen.

be applied, at least in the mouths of persons who exhibit considerable susceptibility to caries, for the purpose of discovering areas on proximal surfaces which are so slightly etched that they probably would not be found either with instruments or radiographs. There is the additional advantage that areas so discovered are immunized at the same time.



FIG. 3A.



FIG. 3B.

FIGS. 3A and 3B. A bicuspid before and after applying silver nitrate, which revealed a slight beginning of caries.



FIG. 3C.



FIG. 3D.

FIGS. 3C and 3D. A bicuspid before and after applying silver nitrate, which revealed that a considerable area of the surface had been slightly etched.

Figures 3A and 3B show the proximal surface of a bicuspid before and after the application of the silver nitrate solution. The first photograph was made a few minutes after the tooth was extracted. It was then dried with a blast of air and the solution was applied with a small pellet of cotton. The silver salts were deposited by applying eugenol. A few minutes later, the second photograph was taken. Careful examination of the two illustrations shows that the blackened areas in Figure 3B are not in the same positions as the high lights in Figure 3A, demonstrating the fact that the high lights are not etched areas. These two begin-

ning decays of the enamel, which have penetrated very slightly, would probably not have been discovered by ordinary methods of examination.

Figures 3C and 3D are corresponding photographs of another bicuspid which presented a smooth surface with very slight discoloration. Visual examination of the tooth, as held in the hand, revealed no definite indication of caries. Figure 3D shows several areas of superficial penetration of the enamel.

Figures 3E and 3F show another bicuspid to which the silver nitrate solution was applied on the proximal surface of the enamel in the same manner as in the preceding illustrations. Evidently there had not occurred the slightest etching of the surface, and the only indication that both illustrations were not made from the same photograph is the slight difference in the high lights.



FIG. 3E.



FIG. 3F.

FIGS. 3E and 3F. A bicuspid before and after applying silver nitrate. This tooth shows no sign of etching.

Figure 3G is a photograph of a temporary cuspid to which the silver nitrate solution was applied soon after it was extracted. The surface of the tooth was perfectly smooth, although it appears rough in the picture as a result of the sharp contrast between the black stain and the high lights.

Figure 3H is a mesio-distal section through a central incisor, showing the penetration of the silver into the carious dentin on the mesial surface, but apparently not quite complete penetration of the enamel of the distal surface.

Figures 3J and 3K illustrate a case in which the application of the solution was made to the mesial surfaces of the upper central incisors of a boy seventeen years of age. This surface was examined by the father, who is a dentist, also by Dr. Prime, previous to the use of the silver nitrate, and neither discovered any suggestion of decay. Figure 3J shows the mesial surface of the right central incisor before, Figure 3K shows it after the application of the solution. Incidentally, the teeth were painlessly slightly separated

with the Ferrier separator, stabilized with modeling compound packed under the bows.

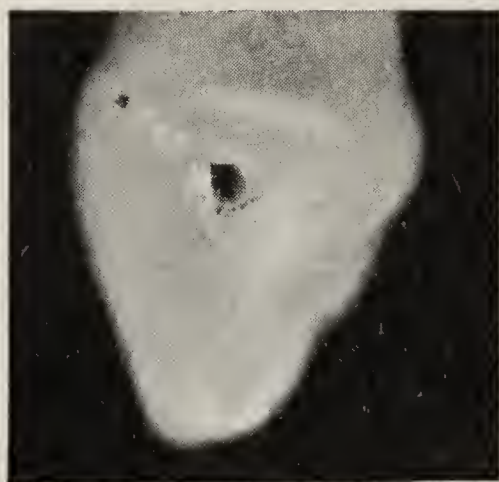


FIG. 3G.



FIG. 3H.

FIG. 3G. A temporary cuspid, the surface of which was smooth and apparently was not decayed. The silver nitrate revealed a considerable surface area that had been etched.

FIG. 3H. A section through a central incisor, made after silver nitrate had been applied to both proximal surfaces. The illustration shows that caries had penetrated the dentin from the mesial surface, but had not fully penetrated the enamel of the distal surface.



FIG. 3J.

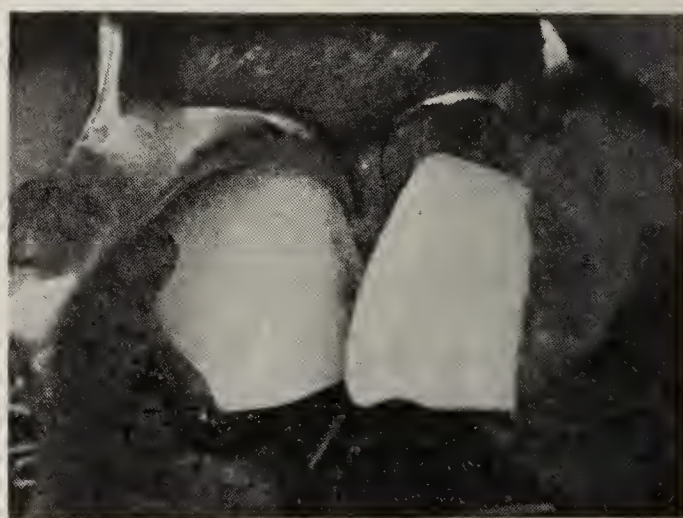


FIG. 3K.

FIGS. 3J and 3K. The mesial surface of an upper central incisor before and after the application of silver nitrate.

Waxed silk floss. In every examination for caries, waxed silk floss should be passed carefully through each contact and then withdrawn occlusally. This should be a part of the regular routine, for the double purpose of testing the tightness of contacts and for the discovery of proximal decays. As the ligature is withdrawn, it should be held hard against the mesial surface of one tooth, and it should then be carried through again and withdrawn with pressure against the distal surface of the adjacent tooth. If the floss drags in passing over the surface, or if it is the least bit frayed, and a decay is not revealed by the instrumental examination or the radiograph, the teeth should be separated sufficiently to make possible an exact diagnosis as to the condition of the enamel. The method of passing the silk through the contact is illustrated in Vol. II, Figure 573.

Transmitted light is also an aid in the detection of proximal decays, as carious areas appear to be of different shade from the surrounding tooth structure. This is not as reliable a test for slight decays as the instrumental examinations or the radiograph.

Illustrations: Volume I, Figures 159 to 171; 197 to 206; 208 to 214.

WHEN THERE IS CONSIDERABLE RECESSION OF THE SEPTAL GINGIVAE, decays will occur at some distance to the gingival of the contact points and should be easily found with the explorers. It should be remembered that practically all decays on the proximal surfaces of the teeth begin just to the occlusal of the margin of the gingivae. When the gingiva has receded only a little, the decay will be to the gingival of the contact, but in cases in which food is forced through weak contacts and the gum is gradually shortened by the impaction of the food, the beginnings of decay follow the gum margin, and may occur in the cementum, if it has been exposed as a result of the recession of the gum.

PROXIMAL DECAYS IN THE INCISORS AND CUSPIDS present the same difficulties in examinations as the bicuspids and molars, yet less in degree, due to the fact that the labio-lingual diameter of the teeth is much less at the level of the contact points. The same methods are applicable.

Illustrations: Volume I, Figures 178 to 183; 207, 208; 215 to 219.

IN CASES OF EXCESSIVE RECESSION OF THE GUMS, which occur occasionally in the mouths of persons considerably past middle age, the cementum may be exposed, particularly on labial and buccal surfaces. Less frequently it will also be exposed on proximal surfaces. If such persons are susceptible to decay, the area involved may be on any surface of the tooth next to the gum, or it may involve all of the exposed cementum.

MARGINS OF RESTORATIONS. The margins of all previously placed restorations should be carefully tested with the explorer point in every position in which the operator does not have a good view of the surface of the tooth while dry. This applies particularly to the gingival margins of both proximal and gingival third restorations. When a restoration projects beyond the contour of the tooth on a proximal surface, overlapping the gingival margin, this may often be discovered in the radiograph, or with the silk floss, which may catch on the projecting restoration.

HYPOPLASIA. ATROPHY. A deformity resulting from a failure of the formation or an imperfect formation of some specific portion of the tooth, and of several teeth together; always the portion of each tooth that was in process of formation or growth at the same time. This deformity is caused by an illness or period of malnu-

trition that interfered with nutrition at the time the particular parts of the teeth affected were in process of calcification.

All four first permanent molars are affected in every case, and in addition the central incisors, or the central and lateral incisors, and, less often, the cuspids also, depending in each case upon the progress of calcification of these teeth at the time of the illness.

The defect generally encircles each tooth, although it is more prominent on the labial surfaces of the anterior teeth, where the enamel is thickest. There may be only a notch in the incisal edge of the incisors — the so-called Hutchinson teeth, or a line of imperfectly formed enamel, often pitted, encircling each tooth at any level from incisal to gingival. The line will always be farther from the incisal edge of the central incisors than the laterals, and always farther from the incisal edge of the lateral incisors than the cuspids. The first molars may have defective spiculae in the positions of the cusps, or a defective ring may encircle these teeth at any level. As a rule, the other teeth are not involved, due to their later formation.

Illustrations: Volume I, Figures 68 to 99.

ENAMEL WHORL. This is a peculiar, defective formation of the enamel in the depth of pits, which occurs frequently in cases of hypoplasia, also occasionally in teeth that are otherwise normal, and in positions in which pits do not ordinarily occur. There is an opening in the surface of the enamel, generally filled with an amorphous material, dark in color, from yellow to black. The dentin is not exposed, although the pits may be deeper than the thickness of the enamel. The dento-enamel junction is depressed and there is a lining of enamel, with the rods in the bottom arranged in a whorl, all pointing toward the center, like the spokes of a wheel. These defects may occur in any position in any tooth.

Illustrations: Volume I, Figures 100, 101.

CORRUGATED TEETH. A rare condition in which the surface of the enamel and the dento-enamel junction are depressed in furrows. There is no relation to a period of malnutrition, as in hypoplasia. The furrows may include the entire crowns of all of the teeth.

Illustrations: Volume I, Figures 102 to 105.

WHITE SPOTS IN THE ENAMEL. White or ashy gray spots occur in teeth which are otherwise of normal color and form. The surface of these spots is usually smoothly glazed. If the area is large, the central portion may not be glazed, but slightly rough. There may be some blotching in the color. The cementing substance is lacking in the outer portion of the enamel in these areas.

Illustrations: Volume I, Figures 106, 107.

WHITE ENAMEL. The enamel is paper white through its entire thickness. There is a failure of formation of the cementing substance; the enamel is very chalky and may be easily penetrated

with an exploring tine. A case is reported in the chapter on hypoplasia in which all of the enamel of all of the teeth was in this condition.

Illustrations: Volume I, Figures 108, 109.

MOTTLED ENAMEL. An endemic deformity, due to the presence of two or more parts per million of fluorine in drinking water, during the time when the enamel of the particular teeth was in process of formation. It is characterized especially by the absence of the cementing substance from between the enamel rods in the outer fourth, more or less, of the enamel. It presents a variety of color in blotches from light yellow to dark brown and black. There are often white areas also. The enamel lacks its normal translucency, although the glaze of the surface is usually complete, except in the cases of very large defective areas. The discolorations are largely confined to the labial surfaces of the upper incisors and cuspids.

Illustrations: Volume I, Figures 15A to 60.

EROSION. A peculiar and very characteristic loss of substance of the teeth, beginning in the enamel, or upon its outer surface, and slowly progressing inward and spreading, gradually destroying and removing the substance of the tooth. At first there are no symptoms except the loss of substance, which looks like a facet that would be left after grinding with a fine stone and polishing. There is no softening of the substance of the tooth, the surface of the affected area being always smooth and polished. After the dentin is involved, there is no line of demarkation in the continuous perfect smoothness of the surface. When the dentin is involved it becomes very sensitive in teeth with vital pulps. This sensitiveness gradually disappears after a variable period of time, due to the fact that secondary dentin is gradually built within the pulp chamber.

Erosion occurs most commonly on labial and buccal surfaces, and generally in the gingival third area. It is rather rare on lingual or proximal surfaces. In a given case, it is generally limited to the surfaces on which it first appears—that is, to labial surfaces, to lingual surfaces, or to proximal surfaces. Erosion appears not to occur except in the mouths of adults. The areas may be dish-shaped, wedge shaped, flattened, irregular or figured.

Illustrations: Volume I, Figures 111-127.

ABRASION OF THE TEETH. Abnormal wear of the masticating surfaces of the teeth, due to unknown causes, is called abrasion. The movements of the teeth in mastication control the form of the abraded surfaces. This unusual wear may be observed in the teeth of persons of all ages, including the temporary teeth. The normal wear of the teeth, apparent in most mouths at the age of forty, should not be classified as abrasion.

Illustrations: Volume I, Figures 131 to 140.

Diseases of the Gingivae and Peridental Tissues

The examination of the gingivae and peridental tissues should always be undertaken with the understanding that in practically every case of disease of these structures, the initial inflammation is in the gingivae and the disease progresses toward the apex of the root. It is therefore of the utmost importance that the slightest inflammations of the gingivae be noted and recorded as a basis for the most effective preventive treatment. This is especially so, in view of the common failure of the peridental membrane to reattach itself to the cementum when it is destroyed by pyogenic infection.

The soft tissues should be examined with good daylight and a mouth mirror or a small lamp inside the mouth. The surface of the gingivae should be free of saliva and should oftentimes be wiped with a pellet of cotton to give the best view.

GENERAL GINGIVITIS OR STOMATITIS. In cases in which there is a general gingivitis or a general stomatitis without an observable local cause, inquiry should be made regarding systemic conditions. See Diseases of the Soft Tissues of the Mouth, also Mouth Manifestations of Skin Diseases and of Systemic Diseases. Particular attention is called to those systemic diseases which involve the blood forming organs.

GINGIVITIS DUE TO DEPOSITS OF SALIVARY CALCULUS. The place of first deposit of salivary calculus is on the *exposed surfaces of the enamel* in contact with the margins of the gingivae, most frequently on the lingual surfaces of the lower incisors, or the buccal surfaces of the upper molars. The initial deposit appears as a thin yellowish-white line on the enamel along the margins of the gingivae. The crests of the gingivae become inflamed and slightly blunted and the normally thin edge is gradually replaced with the deposit. As the process continues, more and more of the gingivae is destroyed and replaced by additional deposit. As the deposit increases, the gingivae become thickened and shortened very slowly. The greater thickness of the tissue gives a broader shelf for the lodgment of more calculus. Each addition to the deposit occurs more or less in layers, between the inflamed soft tissue and the calculus previously deposited. This process may gradually destroy all of the gingivae to the cemental line, and then continue, destroying and replacing with additional calculus, the body of the gingivae attached to the cementum and subsequently the peridental membrane, alveolar process and gum tissue.

The deposit of calculus tends to spread in all directions on the surfaces of the teeth that are not kept clean by the rubbing of food

over them in mastication, or by the movements of the tongue, or by artificial cleaning. As a result the deposit very commonly replaces in form the tissue which is progressively destroyed.

When the deposit is removed, it exposes a red, inflamed surface, which bleeds easily. The irritation of the tissue, caused by the contact of the deposit, offers opportunity for infection, and suppuration occurs from time to time, destroying parts of the tissue and this gives opportunity for more deposit. Occasionally the breath will be foul, but the patient may have no knowledge of the presence of the deposit. The only discomfort to the patient occurs after so much of the supporting tissues have been destroyed that the tooth becomes loose.

As a rule, all of the supporting tissues are destroyed to the level at which the deposit has been laid down. Beyond that level, the attachment of the peridental membrane to the cementum remains intact so that, in cases in which a portion of the peridental membrane has been destroyed, there is no pocket along the side of the root.

Illustrations: Volume IV.

GINGIVITIS DUE TO DEPOSITS OF SERUMAL CALCULUS. The term serumal calculus has been commonly applied to deposits of calculus occurring on the enamel, within the subgingival space, also on the surface of the cementum subsequent to detachment of the peridental membrane. *They never occur on the exposed surface of the tooth.* Clinically, this is a distinct type of calculus, although it does not differ materially from salivary calculus in composition. It accumulates very slowly, and, in cases in which there has been no detachment of the fibres of the peridental membrane from the cementum, when it is deposited on the enamel, it is in the form of small, flat scales or flakes. These deposits are usually not far below the margin of the gingivae and may be observed through the thin tissue as bluish-red spots. In other cases, they may be discovered only with the short, sharp blade of a scaler, which may be carried beneath the gingiva with the edge of the blade in contact with the enamel. When the blade touches the deposit, it may be lifted away from the enamel, carried over the deposit, and again placed in contact with the enamel on the side of the deposit toward the cemental line. There is always a free surface of enamel beyond the deposit.

Illustrations: Volume IV.

DEPOSITS OF SERUMAL CALCULUS ON THE CEMENTUM, in cases in which the peridental membrane has been detached. These deposits are nodular in form, and always occur some time after the peridental membrane has been detached from the cementum at the level at which the deposit is located. These are therefore to be considered a result, rather than a cause of pocket formation,

although they undoubtedly contribute an additional source of irritation within the pocket. The accumulation of this deposit may be extensive, but there is always an exposed surface of cementum between the deepest deposit and the bottom of a pocket. Deposits may be discovered with a scaler with a short, sharp blade, using the technic just described. Deposits on proximal surfaces may be observed in radiographs, but those on buccal, labial or lingual surfaces may not be certainly discovered in a film.

Illustrations: Volume IV.

GINGIVITIS DUE TO LOCAL TRAUMA. The first signs of traumatic inflammation of the gingivae are very slight swelling and discoloration of their margins. The normally thin edge may be slightly blunted, or somewhat rounded and thickened, with the color a little more red than the surrounding tissue. Each such area, no matter how slight, should be recorded and immediate inquiry made as to the cause. The causes may be conveniently divided into four groups:

1. Lack of contact of the teeth, which may result from any of the following: movements of teeth following the extraction of neighboring teeth; abnormal positions of teeth; excessive lateral or protrusive stress; slight weakness of contact without an actual open space; proximal decay; a proximal restoration which does not make contact; the loss in width of a neighboring space, as by a restoration placed without restoring contact, thus permitting either of the two proximating teeth to be moved by the force of occlusion of the teeth of the opposite jaw; and excessive proximal wear of the teeth.

2. Improper contact of the teeth, which may result from any of the following: abnormal forms of teeth or malpositions of teeth which cause the point of contact to be abnormally broad or in an abnormal position; embrasures that are abnormally narrow, due to the flatness of the proximal surfaces of teeth; excessive wear of the proximal surfaces, which changes the points of contact to flattened surfaces of constantly increasing area, that are kept in contact by the mesial movement of the teeth; and the "elongation" of teeth due to the loss of those with which they normally occlude.

3. Margins of cavities of decay and improperly finished margins of restorations, crowns or other appliances which cause irritation of the gingivae. The gum tissue is red and swollen and bleeds on the slightest touch. The patient may complain that the gum bleeds when the teeth are brushed, but there is usually no complaint of pain. Occasionally the inflammation will cause pain.

4. Lack of cleanliness or injuries by the patient, such as lack of cleaning by vigorous mastication; lack of artificial cleaning; injuries with the tooth brush, tooth pick or silk floss.

In every routine examination, all contacts should be tested with silk floss as a part of the examination of the gingivae. In many cases, the too free passage of the contact with the silk will lead to the discovery of inflammation of the septal gingivae which might otherwise have been overlooked.

Illustrations: Volume IV.

GINGIVITIS AND PERICEMENTITIS DUE TO EXCESSIVE OCCLUSAL STRESS. Excessive pressures of opposing teeth of the two arches, that cause either abnormal wear of the occlusal inclined planes, or lateral movements of the teeth, which result in the absorption of the bone of the alveolar process, should be looked for in the examination of the mouths of adults. It should be remembered that excessive pressure on some of the teeth is accompanied by subnormal pressure on others, which is likely to result in loss of tone of the periodontal membranes. Any disturbance of the "equitable distribution of masticating force"* may result in changes in the periodontal structures. There is at hand no sufficiently accurate device for measuring these pressures, and minor stresses can not be satisfactorily determined.

Localized areas of excessive stress may be discovered by testing the occlusion with carbon paper placed between the teeth, while the patient makes lateral and protrusive movements, or by studying models mounted on an articulator, but neither plan is very satisfactory. Lateral movements of teeth under stress are often most accurately determined by holding a finger against the buccal surface of one tooth after another, while the patient holds the teeth in occlusion and makes lateral and protrusive movements of the mandible.

Excessive occlusal stress may result in the gradual migration of a tooth without apparent injury to its supporting structures. There may develop a traumatic apical pericementitis as a result of pressure principally in the line of the long axis of the tooth, with or without absorption of apical bone and without necessarily affecting the condition of the pulp as to vitality. The principal symptom is the tenderness of the tooth to pressure; there may be dull, persistent pain. The tooth is generally not loose. A lateral pericementitis may result from excessive stress on the occlusal inclined planes in lateral movements of the mandible. This will cause inflammation of the periodontal structures, particularly on the side of the root toward which the pressure is directed, and may cause absorption of the bone, while the principal fibres of the periodontal membrane maintain their attachment to the cementum. There may also occur a lateral pericementitis on the opposite side of the root, due to the stretching of the periodontal fibres. The tooth will become loose, but there will be no pocket except as a result of subsequent infection. The protrusive movement of the

*Phrase introduced by George H. Maxwell.

jaw may cause a similar condition of the incisor teeth. In mouths in which many metallic restorations have been made, marks of opposing cusps may indicate surfaces that are too prominent. Undue bucco-lingual movement of a tooth, without detachment of the peridental membrane, is usually an indication of excessive buccal or lingual pressure; also the gradual separation of two teeth, by which the contact is opened, when there is no detachment of the peridental membrane, should always suggest that an opposing cusp is striking too hard between the teeth that have moved apart.

After pockets have formed along the sides of the roots, the tendency of each tooth to move away from the pocket complicates the problem of making a satisfactory diagnosis. Slight inflammation of the gingivae to the buccal or lingual of individual teeth, without apparent local irritation, should call for a study of occlusal stress.

Illustrations: Volume IV.

CHRONIC SUPPURATIVE PERICEMENTITIS. PYORRHEA ALVEOLARIS. This is a slowly progressive non-specific inflammatory process, in which pyogenic organisms play at least an intermittent part, characterized by the detachment of the soft tissue from the surface of the root, thus forming a pocket along the side or sides of the root. The area of alveolar bone adjacent to the pocket is gradually absorbed because it can no longer function, or is destroyed as a result of the inflammatory process. There is the tendency for the tooth to move in the direction away from the pocket, thus disturbing the occlusion, and in cases of pockets on proximal surfaces, to open the contact with resulting lodgment of food debris between the teeth. Pockets progress in depth toward the apex of the root, rather than laterally, and single rooted teeth are particularly liable to become more and more loosened with the increasing detachment of the peridental membrane. Deposits of serumal calculus are likely to occur on the denuded cementum.

The gum margin is usually inflamed and bleeds on slight irritation, as does the pocket tissue, except in cases in which there has been a sufficient downgrowth of epithelium to afford good protection to the connective tissue. A peridental explorer may be passed into the pocket between the root and the overlying tissue. The depth of pockets, measured preferably from the cemental line, should be recorded in millimeters. Pressure on the gum with the finger may reveal the absence of more or less of the alveolar process on the buccal or labial, or lingual side of the root, and pus may be expressed from the pocket. The extent of destruction of bone in the interproximal regions, may be shown accurately with a radiograph, or may be determined with the peridental explorers.

The progress in depth of pockets is usually very slow; two or three years may be required for as many millimeters increase in depth. There may be little or no discomfort, particularly during

the early stages; however, as the pocket becomes deeper, interference with the free discharge of pus into the mouth and a more acute infection in the deeper tissues will be accompanied by considerable pain and tenderness. This may cause the tooth to be slightly elevated in its socket and tender to pressure when the jaws are closed.

In cases in which many teeth are involved, the breath is likely to be foul, there will be a mild ptyalism with drooling on the pillow at night, the cervical glands will be enlarged, and the general health may be impaired.

Illustrations: Volume IV.

LATERAL PERICEMENTAL ABSCESS. In cases of chronic suppurative pericementitis in which the pockets are rather deep, an acute infection may occur in the deeper portion of the pocket and, because of some interference with the free discharge of pus at the gum margin, an acute pericemental abscess may develop. The gum will be swollen, together with other symptoms of acute infection, and slight pressure on the gum may cause intense pain. The tooth will usually be very tender to the touch. If a periodontal explorer is carefully placed in the pocket and the tissue is slightly retracted from the root, pus will be discharged. Such an abscess is in no way related to the death of the pulp; the pulp may be vital.

Illustration: Volume IV.

Diseases of the Dental Pulp and Periapical Tissues

HYPEREMIA of the pulp consists of a sudden expansion of its blood vessels, usually in response to thermal shock. The only symptom is pain, which may be very severe for the moment, then gradually disappear. Hyperemia may be caused by caries of the dentin, by chemical irritation, by hot or cold drinks or food, by breathing through the mouth in cold weather, by heating the tooth with a bur or stone in the dental engine, or by placing a metallic restoration too close to the pulp. The patient's description will usually suffice to make a diagnosis. A test may be made with hot gutta-percha or ice placed in contact with the tooth, or with an electric pulp tester. The teeth adjacent to the suspected tooth should be tested first to determine the approximate heat or cold or amount of current that will elicit a mild response. A tooth with an hyperemic pulp should respond to the same test very promptly with considerable momentary pain. Freedom from pain after severe and often repeated or long continued hyperemia usually indicates the death of the pulp, which occurs from strangulation, as the enlargement of the arteries prevents the blood from escaping through the veins.

ACUTE PULPITIS. Inflammation of the pulp may result from an often repeated hyperemia. During the intermediate stage it may be difficult to make a differential diagnosis. As inflammation develops, the pain may be the same in severity, but will continue for a longer time. Mild or severe inflammation of the pulp may be caused by the extension of caries in the dentin. The pain may be very severe and last for hours. It is likely to be more severe when the patient lies down. If not relieved, it may gradually subside for a time and then return in another paroxysm. Eventually the pulp will die.

A microscopic diagnosis may be made between a serous and a purulent pulpitis; clinically, this cannot be reliably done unless the pulp is actually exposed, and the differentiation in advance is of no particular advantage in treatment.

CHRONIC PULPITIS. In chronic inflammation of the pulp, the patient may experience no pain or discomfort; there may be mild pain occasionally over a period of weeks or months, or reflex pain anywhere in the distribution of the fifth nerve, without any symptom of discomfort about the tooth. When the pulp is partially dead, it may still respond to any one of the tests mentioned. Occasionally in multi-rooted teeth, the pulp tissue in one root may be vital and respond to a test, while that in the other root or roots may be purulent. When metallic restorations in two teeth are in contact, the tooth to be tested should be isolated with the rubber dam before

making the electrical test. In all cases of pulpitis, the vitality of the pulp is eventually destroyed.

DEAD PULP. The death of the pulp may usually be determined by its failure to respond to a thermal or electrical test, although these tests are more reliable in determining the vitality of the pulp by actual response to the test. Sensation may be so reduced by the building of secondary dentin that no response from a remaining vital shread of pulp tissue may be elicited, or the pulp canal may be entirely obliterated by the building of secondary dentin. The first intimation that the pulp is dead may be the discoloration of the crown, or tenderness of the tooth due to an extension of the inflammation into the apical tissues, or the discovery of a radiographic shadow indicating a rarefied region in the periapical bone. A radiograph should be made of every case in which the pulp is dead, to determine, if possible, whether or not the periapical tissues may be involved, and to what extent. The differential diagnosis between simple pulp death and the death of the pulp complicated with destruction of periapical tissues, may be the deciding factor between the conservation and extraction of the tooth.

CHRONIC HYPERPLASTIC PULPITIS. In very occasional cases, after a pulp has been in a state of chronic inflammation for some weeks or months, it retains its vitality and becomes hyperplastic. This usually occurs in cases in which the pulp is widely exposed by caries. The pulp tissue may completely fill the cavity of decay and is usually covered with a thin layer of epithelium. This tissue is only mildly sensitive to slight pressure, and bleeds freely when injured. The bulk of the tissue may be removed with but little pain.

CALCIFICATIONS GROWING FREE IN THE PULP TISSUE. *Nodular calcifications* are generally formed in the bulbous portion of the pulp and most frequently in the molar teeth. *Fusiform or jointed calcifications* occur within the root canals and are usually disposed with their length parallel with the length of the canal. As a rule, these calcifications cause no pain or other symptoms. They may generally be discovered with the radiograph, although it is not always possible to determine whether the calcified mass is free or attached to the dentin wall, unless the pulp chamber is open and the movement of the mass may be detected. If the radiograph shows a little space, apparently occupied by pulp tissue, on either side of the calcified mass, this usually indicates that the mass is unattached.

Illustrations: Volume IV.

CALCIFICATIONS ATTACHED TO PULP CHAMBER WALLS. In cases of abrasion, erosion, slowly progressive dental caries and other conditions in which the dentinal fibrils are irritated, additional dentin may be added to the walls of the pulp chamber. The first building will usually consist of continuations of the dentinal tubules; later the growth changes to a clear calcification, without

tubules. The dentin then becomes insensitive, as the connection of fibrils with the odontoblasts is cut off. The extent of these calcifications may be determined with reasonable accuracy with the radiograph. They cause no pain. It should be recognized that very gradual additions of tubular dentin throughout life may be considered as normal.

Illustrations: Volume I, chapters on Erosion and Abrasion; Volume IV, Pulp Calcifications.

PERIOSTITIS. OSTEOMYELITIS. NECROSIS. See Surgical Disorders.

ACUTE CELLULITIS. LUDWIG'S ANGINA. See Surgical Disorders.

MAXILLARY SINUSITIS. See Surgical Disorders.

TRAUMATIC APICAL PERICEMENTITIS. The periapical tissues may be inflamed as the result of an injury, such as a blow on the upper incisor teeth, or excessive occlusal stress in mastication. In either case the pulp may be vital, although the injury to the periapical tissues may be sufficient to cause its death. The tooth will be tender to pressure, particularly in the direction of its long axis. There may be no pain, or the degree of pain will depend upon the extent of the injury. The radiograph may indicate some destruction of bone, resulting from a severe blow on the tooth, or it may show either slight absorption or condensation of bone in the apical region due to excessive occlusal stress over a considerable period of time.

ACUTE APICAL PERICEMENTITIS. An acute inflammation of the periapical tissues may result from an extension of a pulpitis. The tissues may recover without damage if treatment is instituted promptly; they may become infected and a chronic or an acute periapical abscess may result. In apical pericementitis the tooth will usually be tender to pressure and it may be slightly elevated in its socket. There may be slight pain at first, which will disappear or become more severe, according to the changes which occur in the tissues.

CHRONIC APICAL PERICEMENTITIS, GRANULOMA, CHRONIC APICAL ABSCESS, CHRONIC ALVEOLAR ABSCESS. Periapical infections practically always occur as a sequella of pulp death, by extension of infection through the apical foramen, or from infection of the degenerating pulp tissue via the circulation. These cases may be discovered in any of several stages; (1) proliferative pericementitis in which there is a thickening of the peridental membrane with slight loss of bone; (2) a rarefying osteitis with granuloma, in which the bone is replaced by granulation tissue; and (3) chronic periapical abscess. (See also Periapical cyst.) There may be no clinical symptoms of any of these conditions, although they may have existed for months or years, and the radiograph may be the principal and oftentimes the only means of diagnosis. There may be a

history of injury, of an acute abscess, or of pulp removal and root filling. The tooth may be tender to pressure. Sufficient bone may have been destroyed that palpation with a finger on the gum over the position of the root apex will reveal a very thin outer plate that will give a little under pressure, or a little of the outer plate may be missing, or there may be a sinus discharging through the gum or externally on the face or neck. (After Prinz and Greenbaum.)

Radiographic examination. In cases in which sufficient bone has been destroyed to be definitely recorded on the radiographic film, a diagnosis may usually be made, although a differentiation between granuloma and abscess is not possible unless the presence of pus is demonstrable by exploration through the root canal or through the gum. The outline of the bone cavity is usually somewhat indefinite on the film in cases of granuloma or abscess, and rather definitely rounded when cysts are formed. If the apex of the root appears to be projecting into the cavity, it may be assumed that the peridental membrane has been destroyed over that root area, although an occasional case has been reported in which it has remained intact. In cases in which there is some question whether there has been actual destruction of bone, the best judgment may be formed from observation of the plate of bone to which the fibres of the peridental membrane are attached — the lamina dura; if the white line on the film, parallel and close to the surface of the root, is continuous about the apex, it signifies that the fibres of the peridental membrane are intact, also that none of the bone immediately about the apex has been destroyed. In many cases, radiographs should be made from several different angles in order to observe the largest possible amount of bone about the apex. The line is not always clearly visible about the root ends when conditions are normal, therefore its absence does not necessarily indicate bone destruction.

Sharp steel probe. A sharp steel probe may be passed through a sinus, or in some cases through the gum, into the bone cavity and the extent of the cavity in the bone may be fairly accurately determined. If the root projects into the bone cavity, its contour may be followed with the point of the probe, thus determining the extent to which the peridental membrane has been destroyed. A slight discharge of pus may follow the withdrawal of the probe.

Examination through root canal. An opening through the crown of the tooth may reveal a dead pulp. Pus may be present in the root canal or may discharge into the canal if a broach is passed through the apical foramen. If no pus is discovered in such an examination, or by probing through the gum, a granuloma, rather than an abscess is the more probable condition.

Illustrations: Volume IV.

PERIAPICAL CYST. RADICULAR CYST. ROOT CYST. This condition

may result from a traumatic pericementitis, or from a granuloma following a chronic pericementitis. The proliferation of epithelial cells from the peridental membrane form an inner lining membrane of the granuloma, and secrete a clear fluid. These cysts are painless, although there may be some tumefaction, due to the gradual enlargement of the cyst by the pressure of accumulated fluid within, which may push the bone, teeth and soft tissues out of position. The diagnosis may usually be made from the radiograph, as the outline of the cyst is regular and sharply defined from the surrounding bone. The discharge of cystic fluid through the root canal or through a gum incision is positive evidence. The cyst fluid should be examined for cholesterin crystals.

A periapical cyst may persist indefinitely after the extraction of the tooth, if the cyst wall is not removed. The diagnosis is usually made from the radiograph.

Illustrations* in Volume IV; also Blair and Ivy, Figure 96; Hayes, Figures 296 B and D; Thoma, Figures 155, 156a, 156b.

ACUTE PERIAPICAL ABSCESS. ACUTE ALVEOLAR ABSCESS. Acute abscess is always a sequella of pulp death. It may occur as a primary lesion from infection of the periapical tissues, by extension from the pulp, or as a secondary lesion following a chronic periapical infection of long standing. The tooth will usually be sore and tender to the touch; it may protrude slightly from its socket and it may be loose. All of the symptoms of acute inflammation are present; the pain is usually severe, due to the confinement of the abscess within the bone. The severe pain will continue until the periosteum has been penetrated, usually on the labial or buccal side. The pain will then be less, but will not abate until the pus is evacuated. The temperature may be very high and the pulse rapid. When the pus penetrates the periosteum, the swelling is increased in the immediate area and fluctuation may be detected by palpation. In case an opening through the periosteum is not readily made by the pus, a subperiosteal abscess will form and it may be difficult to diagnose, due to the fact that the pus is confined between the surface of the bone and the tense periosteum. Pus is generally discharged through the gum into the mouth. In the upper jaw, pus may discharge into the floor of the nose or the maxillary sinus, or through the cheek. In the lower jaw, the discharge may be below the mylo-hyoid muscle or may burrow under the deep fascia into the sterno-cleido-mastoid or clavicular region.

The radiograph may show an area of bone about the root apex to have been previously destroyed by a chronic infection. If the acute abscess is a primary infection, insufficient time will have elapsed for destruction of enough of the structure of the bone to show in a radiograph.

Illustrations in Volume IV; also Hayes, 179, 180, 181, 182.

*See statement, page 78, regarding references to illustrations in other books.

Routine Mouth Examination

The routine mouth examination is part of the program of periodic care desirable for all persons, in carrying out the most effective conservation of the dental structures. It should consist of a general survey of the entire mouth, followed by a critical examination for decays and other diseases and abnormalities of the teeth and their supporting structures.

PATIENTS' PHYSICAL CONDITION. As a part of every examination the dentist should have the best available knowledge of the patient's general physical condition. This is important, not only in connection with the management of regular patients who come for routine examinations, but also for those who may present for the first time while suffering, or who may be referred by the physician on account of some systemic effect from a possible local focus of infection. While it is without the sphere of the dentist to make a full physical examination, he may by such observation and inquiry as the circumstances will permit, gain much information which will be of service. The patient's general manner, alertness in movements and conversation, the color of the skin, the facial expression, etc., will usually give the dentist sufficient information to guide him in the conduct of his operations. When there are mouth infections which are evidently of long standing, and particularly if the patient seems not to be in robust health, the dentist should refer the patient to a physician for a thorough physical examination.

In occasional cases, the dentist should ask the patient's physician for a statement of his physical condition.

PERSONAL AND FAMILY HISTORY. The personal history is occasionally of importance in the diagnosis and particularly in treatment planning in operative dentistry. It is often desirable in other dental fields, particularly in oral surgery.

Inquiry into the family history, especially in its hereditary aspects, should be made in relation to congenitally absent teeth, supernumeraries, irregularities and in the tendency to disease of the periodontal membranes and susceptibility or immunity to dental caries.

THE GENERAL SURVEY.

For the general survey, the chair should be tipped about half way back, in order that both the lower and upper teeth and all of the soft tissues of the mouth may be in view for inspection. The lips should be retracted with the fingers to give the best possible view, first of one side of the mouth, then of the other. The mouth mirror should generally be used for this examination and on occasion a mouth lamp. Other instruments are not required.

Four observations should be made and recorded in each case:

The occlusion of the teeth should be noted, and for adults, the general condition of the teeth as to occlusal wear.

The patient's care, as evidenced by the cleanliness of the mouth.

The degree of susceptibility, as indicated by the type and number of decays.

The general condition of the gingivae.

In addition, swellings, tumefactions or inflammations of the soft tissues should be noted for further investigation, and any of these may at once be given precedence over the routine examination. However, as a rule the patient will be conscious of such conditions and will call them to the attention of the dentist for immediate investigation. These will be considered under Special Examinations.

An opinion should be formed during the first examination of each new patient as to the general condition of the mouth and the problems presented in both patient management and treatment; if several teeth are missing, or many of the restorations are large; if several teeth have lost their pulps, or if there are considerable deposits of calculus, there would be no question that the mouth had been neglected, presumably because the patient had never quite understood the possibilities of conserving the teeth. The dentist must, in such cases, make every effort to inform patients to the end that they will give full cooperation.

THE CRITICAL EXAMINATION.

The critical examination and the record to be made as it progresses should include the dentition, dental caries and other lesions of the hard structures of the teeth, diseases of the gingivae and peridental membranes and the diseases of pulps and the periapical tissues.

A definite plan should be followed, although it is not necessary to make a separate survey of the mouth for each item listed. The plan presented herewith will be found to be very satisfactory. It is not important that this particular plan be followed. Any other which covers the field will do as well. It is of the utmost importance, however, that each dentist train himself into the habit of following a regular plan in order that nothing may be overlooked. For the examination of the lower jaw, the chair should be upright; for the upper jaw it should be tipped back.

It is suggested that the teeth of the lower right side should be examined first, beginning with the incisor region and progressing to the third molar, then the teeth of the left side in the same way. The upper right and left sides may then be examined. If the operator will form the habit of standing in the left side in front position, as illustrated in Figure 513, Volume II, for the examination of

the lower left side, it will relieve the muscles which are on greater tension several hours each day in the positions on the right side of the chair.

The examination of each side of each arch may be completed by first using the explorers to locate, in order, pits and fissures, gingival third decays and proximal decays. The silk floss may then be used to discover, if possible, any slight roughness of the enamel near the contacts, and to test the condition of the contacts.

The inspection of the gingivae and gums, followed by the instrumental examination with scalers and peridental explorers should follow the same routine as for caries.

The examination as to conditions of pulps and the periapical tissues should then be made in the same order.

The detail of making these examinations has been given for the various conditions, and the methods of recording each item will be explained and illustrated.

FREQUENCY OF ROUTINE EXAMINATIONS. A complete examination of both the soft and hard tissues of the mouth should be made at regular intervals, the frequency to be determined for each patient. For most patients, this should be four times a year until the first molars erupt, then three times a year during the childhood period, and twice a year thereafter. The arrangement for periodic general care, including the examination, is presented in Volume III, page 2, under the heading "Program for Treatment."

TIME REQUIRED FOR A THOROUGH MOUTH EXAMINATION. In many mouths, there is no more difficult technical procedure in dental practice than a thorough examination of the teeth and the investing tissues. The dentist should never be rushed for time while making an examination. If he recalls a patient twice a year, the patient has the right to expect that he will be thorough in his effort to find decays and inflammations of the gingivae before they have made much progress and the dentist should not disappoint him.

Failure of many dentists to educate their patients to understand the value of mouth examinations, to the end that they are willing to pay a proper fee for the service, has been a great deterrent to thoroughness. Each dentist should be most painstaking in every examination, regardless of the time required, and should charge a just fee.

THE FIRST EXAMINATION. On the occasion of the first examination of each patient, certain special data should be recorded as to the condition of the mouth at the time, including the charting of the restorations then in place; any unusual findings relative to the gingivae and peridental tissues; also the condition of pulps, previous root canal fillings and periapical involvements. As a rule, a full set of radiographs should be made as a part of this examination.

PREVIOUS RECORD. On the occasion of each recall of a patient

for routine examination, the previous examination card, also the permanent record of the patient, and all radiographs on file should be at hand for review by the dentist before the patient is admitted to the operating room. The previous examination card will show the operations performed during the last series of appointments, and these should be checked over as a part of the examination to be certain that they are in good order. This card will frequently have memoranda which were made to call special items to the dentist's attention at this new examination. The permanent record will be at hand for reference as to the previous service and the radiographs will often be consulted in connection with questions that arise.

INSTRUMENTS AND OTHER AIDS IN MAKING EXAMINATIONS. Certain instruments and appliances should always be on the operating tray, and others should be conveniently at hand, for the routine dental examination. Each operator should have at least two sets of the instruments regularly used in making examinations, so that one set may always be ready for use.

INSTRUMENTS. The following instruments should be on the operating tray, or immediately accessible, when the examination is undertaken.

Mouth mirror.

Three explorers, one almost straight, and a pair of right and left curved instruments.

Pair of periodontal membrane explorers.

Cotton pliers.

Cotton.

Gauze rolls.

Silk floss.

Air syringe.

Water syringe, and warm water.

MECHANICAL SEPARATOR. A separator, preferably one of the new Ferrier separators, illustrated in Volume II, Figure 501, will often be required in the examination of the proximal surfaces of the teeth for decays, which may not be discovered by other methods, due to the fact that the surfaces are of such form that a satisfactory examination can not be made without moving the teeth slightly apart.

SHARP STEEL PROBE. A sharp steel probe, very slightly curved toward the point, is of value in determining the different densities of hard structures enclosed within the tissues of the jaws. With this instrument, enamel, cementum, normal bone, softened bone and necrosal bone may be readily differentiated.

RADIOGRAPHIC EXAMINATIONS. Radiographic examination of all of the teeth, with the bone adjacent to the roots, should be made

as a part of the first examination for every adult patient, to locate possible areas of rarefied bone due to absorption, or apical or periodontal infection. Radiographs should be made for children when conditions are present which suggest the possibility of infection, also to observe the state of development of the teeth, etc. Subsequent radiographic examinations should be made of individual teeth or of all of the teeth as may seem desirable in each case. Teeth from which pulps have been removed and root canal fillings made, should be examined radiographically at regular intervals. Unerupted teeth, impacted teeth, supernumerary teeth, odontomes, tumors, bone destroyed by infection, cysts, foreign bodies, fractures and other lesions of the maxillae and mandible may be shown with radiographs.

In connection with pulp treatment. The sizes of pulp chambers and root canals, the extent of calcifications within the canals, measurements of the length of canals and their accessibility to wires placed in them, also to some extent the perfection of root canal fillings, may be determined by radiographs.

Diagnosis of caries. Radiographs are helpful in the early diagnosis of proximal decays. These may be "bite-wing" films which, being taken with the mouth closed, show the crowns and only a part of the roots of several upper and lower teeth on the same film. Films taken of the teeth of each arch separately, which include all of the bone surrounding each root, give an equally good showing of proximal decays.

PULP VITALITY TESTS may be thermal, with ice or hot gutta-percha; electrical, with a current that may be varied in strength by a rheostat or otherwise. The death of the pulp may be determined by percussion when there is inflammation of the apical tissues.

TRANSILLUMINATION TESTS. A lamp placed on the lingual side of either jaw is occasionally helpful in showing abnormalities or areas of inflammation.

A lamp placed in the mouth, with the patient in a dark room and with the lips closed, may reveal the presence of fluid or excessive tissue growth within the maxillary sinus.

ANESTHESIA AS AN AID IN DIAGNOSIS. Whenever it becomes necessary to make an incision, use a probe or otherwise do something that would ordinarily be painful, to gain information essential to a proper diagnosis, an injection of procain may generally be made to anesthetize the region.

Prognosis and Planning of Treatment

In practice, the prognosis and planning of treatment are inseparable, as a choice of plans in treatment must be made on the basis of the dentist's best judgment as to the future of each case.

The treatment to be employed should have special consideration in practically every case that presents, if the best service is to be given. It is also an important factor in securing patient cooperation and in the successful management of a practice. The basis for the planning of treatment consists of a complete and thorough examination, properly recorded — a full pathologic picture, followed by a correct diagnosis. This must be considered with all related data in planning the treatment.

THE AGE OF THE PATIENT has much to do with treatment planning.

During the period of the temporary teeth the principal consideration is usually the management of the child, which will be dependent largely on the development of mutual understanding, to gain the child's interest and cooperation. For young children, operations must be performed in the least possible time and with the minimum of pain, and temporary measures should often be employed to tide over until a more propitious time for permanent service. This must be done in such manner as to enhance the objective of child management without jeopardizing the eventual satisfactory outcome of the service.

During the childhood period of the permanent teeth, the vitality of pulps, necessary to the completion of the roots and therefore to the preservation of the teeth themselves, must have first consideration. This is the period of greatest susceptibility to caries and there is the continued question of child management; also the long view ahead through the many years during which the teeth may be conserved if the service during this period is successful. This is the most critical time for the large majority of patients and the dentist who learns to recognize varying degrees of susceptibility and plans his treatment accordingly, while maintaining the patient's interest, should achieve a high degree of success in carrying patients through to adult life with a full compliment of teeth with vital pulps.

During the adult period, maintenance of the stability of the denture, free from infection which might jeopardize the health, demands increasing consideration with advancing years. The susceptibility to caries is less and the pulp chambers are of smaller

size, as a result of the continued activity of the odontoblasts in building dentin, thus reducing the danger of pulp involvement directly by caries or as a result of the placing of restorations. The teeth are less sensitive and the management of patients presents no major problem. However, the wear and tear, resulting from years of use, gradually transfers attention more and more to the supporting structures. The occlusal surfaces become worn, permitting an extension of the lateral movements of the mandible, with slight loosening of the teeth, which results in the wear and consequent flattening of contact areas. The gingivae and peridental membranes often fail to maintain their tone, due to some constitutional impairment, and gradually weaken their resistance to continuous or frequently recurring irritations. The masticating machine requires close watchfulness, particularly in the matter of readjustments for the protection of the supporting structures.

OTHER CONDITIONS, such as the occlusion, the importance of proper diet, the vigorous use of the teeth in mastication, the estimate of susceptibility, the home care and the education of each patient to an understanding of the health problems involved, in order to secure the best cooperation, are all factors to be considered in treatment planning.

THE SELECTION OF MATERIALS FOR RESTORATIONS is related to all of these matters, and additional limitations are imposed by the esthetic requirements and the personal equation of the operator in manipulating the several materials. The fee and other circumstances peculiar to the patient or the particular operation often require consideration.

THE MOUTH AND THE GENERAL HEALTH. In many cases a study of the mouth conditions, as related to the health of the patient, will determine the general plan to be followed in treatment. Oftentimes, the plan which permits of the best prognosis as to the use of the teeth in mastication, fails to promise the best protection for the patient's health. In such cases, the safe procedure must be in favor of the general health, yet the fact must not be overlooked that the mastication of food is the first essential step in proper nutrition, upon which continued good health depends.

The field of operative dentistry, as outlined in the introduction to this volume, includes all problems related to the occurrence and prevention of chronic mouth infections. The general practitioner assumes responsibility for the conservation of the health of his patients in the planning of treatment. His prognosis as to this or that method must be based on his knowledge of the pathological relationships of technical service for the preservation of the vitality of pulps and of the integrity of the gingivae and peridental tissues. His measure of success in this is his contribution to the protection of the health of his patients throughout their lives.

This brief discussion of problems in the planning of treatment is projected into the clinical treatment of patients in subsequent volumes of this work. Specific cases within the domain of operative dentistry are presented in connection with the discussion of many cases and conditions in Volumes III and IV. The interrelation of the pathology and treatment of dental caries with the diseases and treatment of the pulp and peridental membrane is so evident as to give no justification for their separate consideration in practice.

Record of the Routine Examination

The record of the examination should be conveniently arranged for study in the planning of treatment, for comparison with radiographs, models, photographs, etc., and should be used as a guide in carrying out the treatment. The treatment to be employed may be conveniently entered on the same chart. There should also be provision for memoranda to be carried over to the next examination and for such entries as may be desirable relative to the management of patients. The latter is described in some detail in Volume III, page 4, under the heading of "The Patient Cycle."

The record of the examination should be in such form that every item, either as to the conditions or treatment, should be readily transferable to the day book or to the permanent record of each patient.

THE EXAMINATION CARD.

The advantage of using the smallest practicable card of standard size, that will accommodate all of the desired data, can not be too strongly stressed. The data to be recorded in the examination is of importance and the card should be filed for future reference. As the years pass, cards accumulate and it may eventually become a problem in a dental office to provide sufficient drawer space. The small card is also more convenient to use in the operating room.

The form of the examination record and its use have been a matter of special study by the author for many years, and a plan has been developed by which every disease and abnormality of the mouth may be recorded on a single card with a minimum of time and effort. This plan is a refinement of one presented in the first edition of the Special Dental Pathology in 1915, which has been in use during the past twenty years.

The examination card consists of a diagrammatic chart of the teeth and gingivae, printed on a white card, 4x6 inches in size. It is illustrated in Figure 4. It contains no printing, except that necessary to guide one in making certain entries of a general nature, such as the name, address, etc. in proper places. The entries are very simple and the space is effectively utilized. The chart is printed in light green ink, in order that the markings which constitute the record may be in contrast with the lines of the chart and therefore be more readily distinguished. There is provision for considerable accessory data. The chart of the teeth is so arranged that there is the least likelihood of error in making the entries. Any other examination chart, which affords convenient opportunity to record the necessary data, will answer the purpose.

Fig. 4. Record of Examination without Special Chart as a Guide

This is a very simple plan of recording, although it does not give as complete information as that illustrated in Figure 5. It should be compared with card illustrated in Figure 5, on which the record is entered for the same examination, using the special chart as a convenient guide. Cavities of decay and pulp conditions are entered in black, inflammations of the gingivae and periodontal membrane in red.

ITEMS RECORDED:

Patient's name, address, telephone number and the date.

THE DENTITION:

Lower right third molar impacted (See center oval).

Upper right lateral incisor abscessed; to be extracted.

DENTAL CARIES:

Mesial upper right second bicuspid.

Distal upper right first bicuspid.

Both occlusal pits in upper left first molar.

Gingival third of buccal surface of lower right second molar.

GINGIVAE AND PERIDENTAL TISSUES; INFLAMMATION:

To the buccal of the upper first and second molars on both sides.

Septal gum tissue between upper right first molar and second bicuspid.

[illegible]

To the lingual of lower incisors and cuspids.

A scaling is indicated (see center oval).

Gum resection is indicated for labial and between lower front teeth (see center oval).

PULPS AND PERIAPICAL TISSUES:

Apical abscess, upper right lateral incisor, to be extracted.

Radiograph shows root filling in the upper left lateral incisor.

Pulp is exposed in the upper left first molar.

CROWN AND BRIDGE.

The upper right lateral is to be replaced with a bridge attached to a mesio-linguo-distal inlay in the cuspid.

The upper left lateral is to receive a porcelain jacket crown.

GENERAL INFORMATION REGARDING THE PATIENT. Space is provided for the following data on the upper part of the card: Name, patient number, address, residence and office telephone numbers, date, age (date of birth for children), number of the examination, time for the next recall, and the classification of the patient.

Patient number. Many dentists number patients consecutively for the particular purpose of keeping a record of the number of new patients during each year. This indicates the progress in practice building. To do this conveniently, a hundred or more cards should be numbered in advance and the cards so numbered should be used only for new patients.

The age is of particular importance in the analysis of service records. For children, it is desirable to record the exact age, which may be most conveniently done by noting the date of birth, provision for which is made on the children's card. For adults, the approximate age will generally be sufficient.

The number of the examination, entered on the card for each patient, enables one to determine the frequency of examinations over any given period of time, and affords opportunity to study the results of service with relation to the frequency of recalls.

The time for recall for the next examination may be entered, and this should be preferably in red ink. This card may then be filed behind a monthly guide card as a reminder to notify the patient when the time arrives.

Classification of patients. Some dentists classify patients as A, B, C, etc., according to their estimate of the desirability of the individual as a patient, considered from all angles; interest, co-operation, punctuality in keeping appointments, promptness in payment, etc. The letter may be entered on the line below the age.

THE GENERAL SURVEY of the mouth may be recorded by special notations in the central oval space. There is also provision, at either end of the oval, for notations as to the occlusion, the patient care, the degree of susceptibility to dental caries, and the general condition of the gingivae.

THE CRITICAL EXAMINATION is entered on the chart for the teeth, where, for each tooth, separate spaces are provided in which to record (1) decays and other lesions of the hard tissues of the teeth, (2) inflammations of the gingivae and peridental membranes, and (3) conditions of pulps and the periapical tissues.

THE CHART FOR THE TEETH AND GINGIVAE is arranged in the form of an oval; the median line of the mouth is vertical, the line between the upper and lower teeth is horizontal. The upper teeth are designated by the figures 1 to 8 — central incisor to third molar, to the right and to the left from the median line; the lower teeth are similarly designated by capital letters A to H. The cuspids

and first molars have heavy outlines to distinguish them from the other teeth and thus reduce the possibility of marking the wrong tooth. The diagram for each tooth provides separate spaces for charting decays of various classes, for conditions of the investing tissues; for conditions of pulps and the periapical tissues; also spaces for recording diseases (other than caries) of the hard tissues of the teeth, abnormalities, supernumeraries, etc. The recording of these will be explained in the following pages.

The figures and letters which indicate the individual teeth are of special importance in entering the record in the day book and in posting it to the patient's permanent record card. Each figure or letter always indicates the same tooth on either side of the arch. In the upper arch the figures 1 and 2 represent the first and second incisors and 6 the sixth year molar, while in the lower arch, C represents the cuspid and F the first molar. These key numbers and letters thus have a special significance which minimizes errors in recording.

ACCESSARY DATA IN CONNECTION WITH DIAGNOSIS AND TREATMENT. Space is provided for the following memoranda on the lower part of the card: Bite-wing films on file, full set of radiographs, case history, models, photographs, previous service checked, estimate of fees, report on case filed, special instruction to patient and memoranda to be noted at next examination. In each instance a cross is made by marks between the parallel lines, to indicate the item.

A report filed is usually a report to a physician who has referred the case to the dentist, with a request that an examination be made for foci of infection. This entry may, however, refer to any other form of report.

Previous service, marked with a cross, is a record that a check up on the condition of previous operations was made at the time of the examination.

Estimate, marked with a cross, indicates that an estimate of the fees for the restorations, treatments, etc. is entered on the reverse side of the card.

Patient instruction, indicates that directions were given to the patient as marked elsewhere on this card. It reminds one to look for the notation. The item may be written in the central oval space, or indicated on the chart of one or several teeth.

Special memoranda may be entered in the central oval space, such as the diagnosis of special conditions, and notations regarding a particular case, as the pulse, temperature, etc.; memoranda of special instructions to the patient, or to be carried forward to the next examination; also the name of the person who referred the patient.

Recording the Examination

The record should be made as the examination progresses. The examination should be so thorough and the record so complete that the card will contain all information necessary for planning the treatment, for making an estimate of fees when desired, and may also be used as a guide in carrying out the various details of treatment. Notations should also be made regarding accessory data that may be of value in the future management of the case. Many rather minute details should be noted, and the entries must be made in an orderly manner, yet without requiring an appreciable amount of time.

Two plans of recording the examination are presented. A very simple plan is illustrated by the entries on the card in Figure 4. In this cavities of decay are marked in black, inflammations of the gingivae in red and conditions of pulp are indicated by abbreviations. Such a record may be made with black and red inks or pencils. The important difference between this plan and that very commonly used consists of the definite record of each inflammation of the gingivae, which must be made if one is to achieve a reasonable measure of success in the prevention of the more serious infections of the peridental structures.

The second plan, illustrated by the entries on the card in Figure 5, is presented for the dentist who desires to make a more complete record, which will be helpful in diagnosis and in the planning of treatment. While the latter plan appears to be more complicated, because of the many items listed on the accompanying chart, it is in reality quite as simple as the former.

To demonstrate the fact that there is really no difference of consequence in the recording, exactly the same conditions are recorded on the two cards in Figures 4 and 5. In the legends accompanying the two illustrations, the interpretations of the markings demonstrate clearly the difference in the information which is recorded by the two methods. Particular attention is called to the fact that one may make use of as few or as many items on the chart accompanying Figure 5 as may suit his own convenience. As a matter of fact, one will find that comparatively few of the items are frequently used; the majority are needed only very occasionally.

CHART TO BE USED AS A GUIDE. To facilitate the entire procedure of making and recording the examination, a special chart has been arranged on which are listed all of the lesions of the teeth and supporting structures for which diagnostic data have been presented in the preceding pages, also a number of other conditions

Fig. 5. Record of Examination with Special Chart as a Guide

This should be compared with the card illustrated in Figure 4, on which the record is entered for the same examination on the plan generally used, except that inflammations of the gums are marked in red.

ITEMS RECORDED: Patient's name and number, address, telephone numbers, age 30, a "B" patient (see classification of patients, page 61). Date 3-12-33, first examination. Recall in Sept. '33. Referred by W. L. Masters.

GENERAL SURVEY: Occlusion DDP, distal relation of both lower first molars, protrusion upper incisors; patient care poor; susceptibility to caries slight; gingivae in fair condition.

THE DENTITION: Upper right third molar missing; lower right third-molar impacted; upper right lateral incisor to be extracted on account of a chronic abscess.

DENTAL CARIES: Etched enamel, buccal of upper right second molar, to be immunized with silver nitrate. Mesial upper right second bicuspid; distal upper right first bicuspid; both occlusal pits in upper left first molar; gingival third of buccal surface of lower right second molar.

GINGIVAE AND PERIDONTAL TISSUES: Inflammation of gingivae between upper right second bicuspid and first molar on account of lack of contact of restoration (26), pocket 4mm. on distal second bicuspid and 3mm. on mesial first molar with deposits of serumal calculus on both roots (19). A mesio-occlusal restoration is to be placed in the first molar to re-establish the contact.

Deposits of salivary calculus on lingual of lower incisors and cuspids (18), pockets varying from 3 to 7mm. on proximal and labial surfaces of these teeth. The labial and septal gum tissue is to be cut away to eliminate the pockets. Lower left central incisor loose (46). Failure to properly brush buccal surfaces of

upper molars (62) and patient was given instruction (see + near lower right corner of card.)

PULPS AND PERIAPICAL TISSUES: Upper right lateral, chronic abscess (C), to be extracted (X); upper left lateral, root filling (R); upper left first molar, exposed by caries (E).

CROWN AND BRIDGE: Upper right cuspid, mesio-lingual distal inlay, with lateral dummy attached, rest on lingual

of central incisor; upper right lateral incisor, porcelain jacket crown.

OPERATIONS BY PREVIOUS OPERATORS (Entered in red): Occlusal both upper second molars; mesio-occlusal upper right first molar; mesial of both upper central incisors; mesio-distal-occlusal lower left first molar.

A full set of radiographs was made and an estimate was made of the fees. See marks in lower section of card.

Rohland, James A *6172, S. Huron St*
Chicago
 NAME ADDRESS
 NUMBER 1678 TEL. North 3142 NPIR
 BUS. TEL. Main 2621
 EXAM. NO. 1
 DATE 3-12-33
 NEXT EXAM Sept '33
 AGE 30
 SEX B
 RACE C
 OCCCLUSION DDP
 PATIENT CARE 123
 REFERRED BY W. L. Masters
 REPORT TO
 CRIES 123
 GINGIVAE 123
 PULPS AND PERIAPICAL TISSUES: Upper right lateral, chronic abscess (C), to be extracted (X); upper left lateral, root filling (R); upper left first molar, exposed by caries (E).
 CROWN AND BRIDGE: Upper right cuspid, mesio-lingual distal inlay, with lateral dummy attached, rest on lingual of central incisor; upper right lateral incisor, porcelain jacket crown.
 OPERATIONS BY PREVIOUS OPERATORS (Entered in red): Occlusal both upper second molars; mesio-occlusal upper right first molar; mesial of both upper central incisors; mesio-distal-occlusal lower left first molar.

CHART TO BE USED AS A GUIDE IN RECORDING ROUTINE EXAMINATIONS

GENERAL SURVEY. Record Occlusion, Patient Care, Estimate of Susceptibility to Caries, Condition of Gingivae.

THE OCCLUSION. See page 68.

First letter for molar relation right side.
Second letter for molar relation left side.
Third letter for incisors, upper or lower.
Use dash if for lower incisors.

NNN Normal.
NNI Molars normal, incisors irregular.
DDP Bilateral distocclusion, up. inc. protruding.
DNP Unilateral distocclusion, up. inc. protruding.
DDR Bilateral distocclusion, up. inc. retruding.
NDR Unilateral distocclusion, up. inc. retruding.
MM-P Bilateral mesiocclusion, low. inc. protruding.
MN-P Unilateral mesiocclusion, low. inc. protruding.

THE DENTITION. See page 69.

/ Congenitally absent.
— Missing (not erupted; extracted).
= Unerupted and overdue.
≡ Impacted.

Delayed eruption, enter age in lingual rectangle.

Temporary teeth retained. Circle with arrow indicating position in arch.

SUPERNUMERARY TEETH. See page 69.

○ Cone.
● Truncated cone.
Y Dichotome.
└ Fused teeth.
= Gemma.

Extra teeth of normal form. Locate on chart, name in center oval.

DENTAL CARIES. See page 70.

○ Pit decay.
() Proximal decay, mesial and distal.
C Gingival third decay, upper and lower.

DYSTROPHIES and other conditions of Hard Tissues. See page 73.

Enter numbers in labial-buccal rectangle.
91 Hypoplasia. Atrophy.
92 Enamel whorl.
93 Corrugated teeth.
94 White spot in enamel.
95 White enamel.
96 Mottled enamel.
97 Erosion. Locate areas in **Blue**.
98 Abrasion. Locate areas in **Blue**.

GINGIVAE; PERIDONTAL TISSUES. See page 71.

Enter numbers as illustrated in Fig. 6C.

16 Diastema, no inflammation.
17 Recession, no inflammation.
18 Salivary calculus.
19 Serumal calculus.

GINGIVITIS DUE TO

LACK OF CONTACT OF TEETH:

21 Separations following extractions.
22 Abnormal positions of teeth.
23 Movements caused by excessive stress.
24 Weak contact, no decay.
25 Proximal decay.

26 Proximal restoration.

27 Loss in width of neighboring space.

28 Detachment peridental membrane.

IMPROPER CONTACT OF TEETH:

31 Abnormal forms of teeth.
32 Malpositions.
33 Embrasures abnormally narrow.
34 Excessive proximal wear.

DEVIATIONS FROM NORMAL CONTOUR:

36 Sharp edges of cavities.
37 Imperfect margins of restorations.

EXCESSIVE OCCLUSAL STRESS:

41 Migration of tooth, no pericementitis.
42 Apical pericementitis, no bone absorption.
43 Apical pericementitis, bone absorption.
44 Lateral pericementitis, bone absorption, no detachment.
45 Lateral abscess.
46 Tooth loose; 45— slightly loose; 45+ very loose.

CHRONIC PERICEMENTITIS. PYORRHEA.

Enter depth of pockets in mm. on sides of root where detachment has occurred.

CEMENTUM.

51 Hypercementosis.
52 Apical resorption of root.
53 Lateral resorption of root.

PATIENT CARE.

61 Lack of cleaning by mastication.
62 Lack of artificial cleaning.
63 Injury in cleaning.

PULPS; PERIAPICAL TISSUES. See page 71.

Enter letters as illustrated in Fig. 6C.

H Hyperemia.
NE Normal pulp, accidental exposure.
E Exposed by caries, vital.
AI Acute inflammation of pulp.
CI Chronic inflammation of pulp.
HP Hyperplastic pulpitis.
PD Partially dead.
D Dead.
NC Nodular calcification.
FC Fusiform calcification.
SD Secondary dentin excessive.
R Root filling.
PR Partial root filling.
T Traumatic apical pericementitis.
AP Acute apical pericementitis.
G Granuloma, chronic apical abscess.
S Sinus from apical abscess.
PC Periapical cyst.
A Acute periapical abscess.

X **RADIOGRAPHS**, single teeth, enter in **Red**.

TREATMENT. See page 74.

PREVENTIVE, for caries, enter same as caries, in **Blue**.

RESTORATIONS:

● Pits.

() Mesial and distal.

(+) Mesio- and disto-occlusal, mesio- and disto-incisal.

C Gingival third, upper and lower.

○ Crown.

Bridge, fixed, to crown and inlay.

Bridge, attached to crown, rest on inlay.

Bridge, removable, pin and sleeve.

Bridge, removable, clasps.

Partial; pin and sleeve, clasp and rest.

Full dentures, enter in center oval.

GUM RESECTIONS:

Labial or lingual.

Interproximal.

Labial or lingual and interproximal.

Labial, lingual and interproximal.

X TEETH TO BE EXTRACTED.

PREVIOUS OPERATIONS. See page 75.

Same symbols as above, in **Red**.

which do not require special discussion, but are necessary to the completeness of the record. The items on the chart are classified under several headings and arranged for convenient reference. Accompanying each item is a symbol, figure or letter, by which it may be recorded in the proper place on the card. By this plan, any item may be quickly recorded by the dentist himself or by the assistant, as the examination proceeds.

The items on the chart are classified under six headings: the occlusion; the dentition; caries and other diseases of the hard tissues of the teeth; diseases of the gingivae and peridental tissues; diseases of the pulp and periapical tissues; and the treatment to be employed. The directions for making the record of each condition are given on the chart and selected items are illustrated in Figure 5. Further illustrations of the entries to be made in recording the various conditions are presented in Figures 6A, 6B, 6C, 6D and 6E.

Examination cards, with average recordings for a variety of patients of different ages will be found in Volume III, Figures 82, 83, 86, 104, 121, 134, 157, 170, 176 and 247.

Gross, Martha 1231 W. State St
 NAME NUMBER 1422 RES. TEL. Maple 673 NPIR ADDRESS Chicago, 4
 BUS. TEL. Main 3421 EXAM. NO. 4
 DATE OF BIRTH 3-5-19 DATE May 10, 1927
 AGE 8 NEXT EXAM Sept '27

RIGHT LEFT

OCCLUSION N 123 PATIENT CARE DN M NP ND 123 CINGIVAE

REPORT TO Mr. John M. Gross 318 Ayers Bldg

PR EV SER VICE ES TI-MA TE

REP ORT PATIENT ME MO
 FIL ED INS TR. FOR WD

FIG. 6A. Examination card for the temporary teeth.

SPECIAL CHART FOR THE PERIOD OF THE TEMPORARY TEETH. See Figure 6A. This has provision for entry of the same data as men-

tioned for the permanent teeth, including the occlusion. The upper teeth are designated by Roman numerals, and the lower teeth by small or "lower case" letters. The diagrams for the cuspids have heavier outlines than those for the other teeth. During the transition period, when the temporary teeth are being shed, one may, at each examination, mark as missing all of the temporary teeth which have been shed, also the permanent teeth which have not erupted, thus recording the existing dentition on each occasion. In the chart shown in Figure 6A, for a child 8 years of age, the permanent first molars and incisors have erupted and temporary incisors have all been shed, as shown by the lines indicating the missing teeth.

The entries for caries and other conditions on the child's chart are made in the same manner as on the chart for the adult. In Figure 6A, the following items are recorded: General survey; the occlusion is normal, the patient care poor, susceptibility high, and the gingivae are slightly inflamed. The patient was instructed in the care of the mouth (see item marked near lower right corner of card), special attention being called to the first permanent molars, the buccal surfaces of which were not being properly brushed (62). There are decays in the temporary teeth as follows: In the occlusal surface of the lower right first and the lower left second molars, also in the distal of the upper right first molar and the mesial of the second molar. In the permanent teeth, decays are marked in the occlusal surface of both lower first molars. The patient's next examination was arranged for "September, 1927."

THE REVERSE SIDE OF THE EXAMINATION CARD provides for the entry of estimated minimum and maximum fees for the operations recorded on the examination chart, also for a memorandum of the arrangement, if any, that is made relative to the terms of payment. There are also several lines on which to enter memoranda of time devoted to incompleted service, as time required for preparing a cavity and making a wax model for an inlay, which is to be added to the time subsequently required for the casting, fitting and cementing of the inlay. The total time may then be entered in the day book as a single item, along with the fee.

RECORDING THE GENERAL SURVEY

The occlusion, the patient care, the estimate of susceptibility to caries and the general condition of the gingivae should be noted and recorded in the spaces provided at either end of the inner oval. On the card shown in Figure 5, the occlusion is indicated as class 1, division 1, the patient care poor, the susceptibility slight, and the condition of the gingivae fair. The three letters indicating the occlusion are entered in the space reserved for the purpose. The other conditions are indicated by marking the figure 1 for good, 2 for medium and 3 for poor.

RECORDING THE OCCLUSION.

SEE DIAGNOSIS, PAGE 25.

The relation of the right lower first molar to the upper should be recorded first, by marking the letter N for normal relation, D for distal relation or M for mesial relation, in the space provided on the card, opposite the lower first molar. The record of the relation of the left lower first molar to the upper should then be similarly entered. The condition of the upper incisors should be indicated as

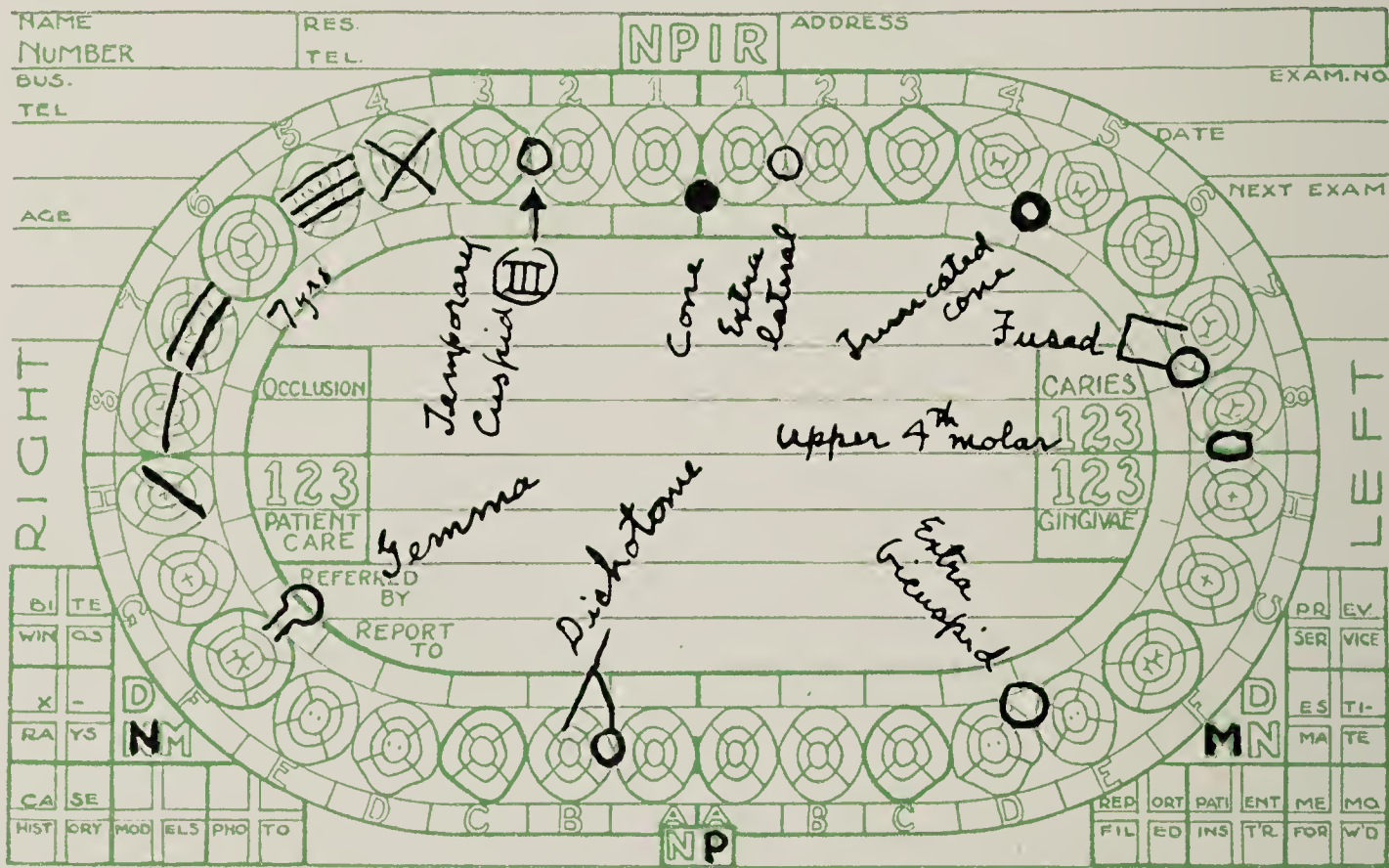


FIG. 6B. Exhibit of recordings of the dentition, including the various forms of super-numerary teeth. The illustration is about two-thirds the actual size of the card.

normal, protruding, irregular or retruding, by marking the proper letter to the labial of the upper incisors. Similarly, the lower incisors, instead of the uppers, may be marked as normal or protruding.

The three letters should then be entered on the card, in the space provided to the left of the oval, with a dash before the letter P, if it applies to the lower incisors. On the chart, Figure 5, the entry DDP indicates that the lower molars are in disto-occlusion on both sides, and the upper incisors are protruding. In Figure 6A, on the chart for the temporary teeth, also on the chart for the permanent teeth, the relation in all three positions is marked normal. In the chart, Figure 6B, the letters N, M and P indicate normal molar relation on the right side, and mesial relation of the lower molar on the left side, with protruding lower incisors. This would be written NM-P in recording the occlusion.

RECORDING THE DENTITION.

SEE DIAGNOSIS, PAGE 27.

It is desirable in connection with the planning of treatment that the exact status of the dentition be recorded, although it is not necessary that this be done at every examination for each patient.

Teeth congenitally absent may be recorded with a diagonal line. See the lower right third molar in Figure 6B.

Missing teeth, those not yet due to erupt, temporary teeth recently shed and teeth previously extracted may be recorded with a mesio-distal line. See the upper right third molar.

Unerupted teeth that are overdue may be recorded by two mesio-distal lines. See the upper right second molar.

Impacted teeth may be recorded with three mesio-distal lines. See the upper right second bicuspid.

Delayed eruption may be recorded by entering the age at which eruption occurs, in the space to the lingual of the tooth chart. See the upper right first molar, erupted at 7 years.

Temporary teeth retained beyond normal shedding time may be recorded by drawing a circle about the number or letter representing the temporary tooth, with arrow indicating its position in the arch. The card shows the upper right temporary cuspid retained between the permanent lateral and cuspid.

Teeth to be extracted should be marked with a cross of diagonal lines. See the upper right first bicuspid.

SUPERNUMERARY TEETH. See illustrations, Figures 2A to 2S. Although supernumerary teeth are not frequently observed, it is desirable that they be recorded and they are therefore included in the items listed on page 65.

Cone. This form should be entered as a circular area in the position it occupies in relation to the other teeth. See supernumerary located in lingual embrasure between upper centrals in Figure 6B.

Truncated Cone. Conical form with flat end instead of point; should be entered as a circle in the proper position. See supernumerary located in lingual embrasure, between upper left first and second bicuspid.

Dichotome. This type consists of one supernumerary and one normal enamel germ, which have become fused together, forming one tooth with a common pulp and root canal. It may be recorded with a Y as in the case of the supernumerary joined with the lower right lateral incisor.

Fused teeth. Formed from two distinct teeth, a supernumerary and a normal tooth, the roots of which are united by cementum. Such a case may be recorded by indicating the position of the supernumerary and drawing two parallel lines with a base line connecting their ends. See the supernumerary fused to the upper left second molar.

Gemma. An enamel bud developed from a supernumerary enamel organ which is entirely separate from the enamel of the crown of the normal tooth to which it is attached. Both have a common pulp. Such a case may be entered by a circle attached to the normal tooth by two parallel lines. See the gemma attached to the lower right first molar.

Extra teeth of normal form. The location of such teeth should be indicated with a circle, and the type of the tooth should be written in the center oval on the card. See the extra upper left lateral incisor, also an upper left fourth molar and a third lower left bicuspid.

NAME		RES.		NPIR		ADDRESS			
NUMBER		TEL.						EXAM. NO.	
BUS.								DATE	
TEL.								NEXT EXAM	
AGE									

RIGHT		LEFT	
123 PATIENT CARE		123 GINGIVAE	
REFERRED BY REPORT TO			
18 18 18 18 18 C 44 46		18 18 18 18 18 C 44 46	
D C B A A B C NP		D C B A A B C NP	

FIG. 6c. Exhibit of recordings of dental caries, of inflammations of the gingivae and periodontal tissues, and of diseases of the dental pulp and the periapical tissues. The illustration is about two-thirds the actual size of the card.

RECORDING DENTAL CARIES.

SEE DIAGNOSIS, PAGE 31

The simple symbols which are in general use — circles and curved lines, are marked in the proper positions on the tooth chart. The center area of each tooth chart represents the occlusal surface or incisal edge, while the four spaces immediately surrounding the central area are for recording decays on the mesial, distal, labial or buccal, and lingual surfaces. See Figure 6c.

The following decays are recorded on the card in the accompanying illustration.

Upper right second molar; buccal pit, and gingival third on buccal surface.

Upper right first molar; both occlusal pits, also pits in the buccal and lingual surfaces.

Upper right second bicuspid; occlusal fissure, and gingival third on buccal surface.

Upper right first bicuspid; distal pit in occlusal surface, and mesial surface.

Upper right lateral incisor; lingual pit, and mesial surface.

Upper right central incisor; distal surface.

RECORDING DISEASES OF GINGIVAE AND PERIDONTAL MEMBRANES.

SEE DIAGNOSIS, PAGES 40-45.

All of the conditions of the gingivae and peridental membranes which may be recorded on the examination card are listed on the chart which accompanies Figure 5. These correspond to the diagnostic data presented in the text. Only a few samples need be given here to illustrate the plan of recording. It will be noted that figures are used to indicate the several conditions in this group. Figures may be placed in the outer circle of each tooth chart to indicate an inflammation and its cause on the labial, lingual, mesial or distal surface of each tooth, or to record the depth of a pocket. A figure placed in the labial, or buccal, embrasure indicates an inflammation of the septal tissue and the condition of the contact.

The following conditions are recorded on the card in the accompanying illustration, Figure 6C.

- 16, between the upper central incisors, indicates a diastema, no inflammation.
- 17, on the labial surface of the upper left cuspid, indicates a recession of the supporting tissues, no inflammation.
- 18, on the lingual surfaces of the lower incisors and right cuspid, indicates deposits of salivary calculus.
- 19, on the lingual of the upper left first bicuspid, indicates a deposit of serumal calculus.
- 21, between the lower left first molar and second bicuspid, indicates the separation of these teeth following the extraction of the second molar, which is marked missing. There is a pocket 4 mm. deep on the distal surface of the bicuspid and a pocket 3 mm. deep on the mesial surface of the first molar, and the figure 19 indicates a deposit of serumal calculus on both roots. A mesio-occlusal restoration is to be placed in the first molar to re-establish the contact.
- 23, between the upper left cuspid and first bicuspid indicates a gingivitis due to the separation of these teeth, the separation being caused by the excessive stress of the cusp of the lower first bicuspid.
- 26, between the upper left second bicuspid and first molar, indicates a gingivitis due to the failure of a proximal restoration to make contact. A restoration in the mesio-occlusal of the first molar is to be replaced to re-establish the contact.
- 28, between the lower right first and second bicuspid, indicates the separation of these teeth due to pyorrhea pockets .5 mm. deep on the distal of the first bicuspid, 7 mm. deep on the mesial of the second bicuspid. The figure 45 records a lateral abscess on the buccal side of the second bicuspid, the pocket being 6 mm. deep. The figure 46 indicates that this tooth is loose. The X is a notation that the tooth is to be extracted.
- 33, between the upper left first and second molars, indicates that the embrasures are too narrow, due to the flatness of the proximal surface contours.
- 37, on the distal surface of the upper left first bicuspid, indicates a gingivitis due to an imperfect margin of a restoration.
- 44, on the buccal surface of the lower left first bicuspid, indicates a lateral periodontitis with bone absorption due to excessive lateral stress. The figure 46- indicates that this tooth is slightly loose.
- 62, on the buccal surface of the upper left first and second molars, indicates lack of proper brushing by the patient. In the lower right section of the card, the item "patient instruction" is marked to record the fact that the patient's attention was called to the failure to brush these areas. This also reminds the dentist to examine these areas on the occasion of the next recall.

RECORDING DISEASES OF THE PULP AND PERIAPICAL TISSUES.

SEE DIAGNOSIS, PAGES 46-50

There are many conditions of the pulp and periapical tissues that the dentist may wish to record, which require no description, or special diagnostic statement. These are listed on the chart which accompanies Figure 6c. A few of the more common conditions will be mentioned in order to illustrate the method of recording. All of these conditions are noted by letters in the rectangular space to the lingual of each tooth. In each case, the letter or letters indicate the condition to be recorded; they are the initial letters of the words used to name the condition. The letters and the list of conditions sufficiently explain themselves.

The following conditions are recorded on the card in the accompanying illustration:

E, upper right lateral incisor, indicates that the pulp is vital and exposed by caries.

R, upper left lateral incisor, indicates a good root filling, also that the periapical bone appears to be in perfect condition in the radiograph.

NC, upper right first molar, indicates a nodular calcification in the pulp chamber.

A, lower right first molar, indicates an acute periapical (alveolar) abscess.

C, lower left cuspid, indicates a granuloma or a chronic periapical abscess, and the X records the fact that the tooth is to be extracted.

NAME _____ RES. _____ ADDRESS _____
 NUMBER _____ TEL. _____ NPIR _____
 BUS. _____ EXAM. NO. _____
 TEL _____ DATE _____
 AGE _____ NEXT EXAM _____

RIGHT

LEFT

BI **TE** **WIN** **QS** **X** **-** **D** **F** **RA** **YS** **NM** **CA** **SE** **HIST** **ORY** **MOD** **ELS** **PHO** **TO**

PR **EV** **SER** **VICE** **ES** **TI-** **MA** **TE** **D** **MN** **REP** **ORT** **PAT** **ENT** **ME** **MO** **FIL** **ED** **INS** **TR** **FOR** **WD**

OCCLUSION **123** **PATIENT CARE** **REFERRED BY** **REPORT TO** **CARIES** **123** **GINGIVAE** **123**

Hypoplasia at 2 yrs
Mottled enamel - light yellow
Erosion - wedged shaped

NP

FIG. 6D. Exhibit of recordings of the lesions of the hard tissues of the teeth, except caries. The illustration is about two-thirds the actual size of the card.

RECORDING LESIONS OF THE HARD TISSUES OF THE TEETH,
OTHER THAN CARIES.

SEE DIAGNOSIS, PAGES 38-39.

In recording the lesions of the hard tissues, one of a series of numbers may be entered in the rectangle to the labial or buccal of each tooth involved, with such additional memoranda in the center oval or elsewhere as may be desired in the particular case. All of the lesions of this group are marked on the card in Figure 6D. It would be an unusual case, however, in which more than one of these conditions would be found in the same mouth.

91. HYPOPLASIA. The number, entered to the buccal of the four first molars and to the labial of the eight incisors on the card in Figure 6D indicates the teeth which show hypoplastic defects, and in the center oval the entry "Hypoplasia at 2 years" records the age at which the illness or period of malnutrition occurred. This is estimated from the position of the defects in the teeth. See the diagram, Vol. I, Figure 79.

92. ENAMEL WHORL. The number is entered to the buccal of the lower right cuspid, and the location of the defect is marked on the labial surface of the tooth in the diagram.

93. CORRUGATED TEETH. The number is entered to the buccal of the lower left third molar to indicate this condition.

94. WHITE SPOTS IN THE ENAMEL. The number is entered to the labial of the upper left cuspid, and the location of the white spot is marked on the labial surface of the tooth.

95. WHITE ENAMEL. The number is entered to the buccal of the upper right third molar; no mark is made on the tooth chart as the enamel of the entire tooth is white.

96. MOTTLED ENAMEL. The figure is entered to the buccal of all four second molars, and all of the bicuspids, indicating that the person lived in an endemic area from about the fourth to the eighth year. The color of the mottled areas and the age are entered in the center oval.

97. EROSION. The figure is entered to the labial of the upper right cuspid and the blue mark indicates that the eroded area is on the labial surface of the tooth. Blue is used to differentiate erosion from caries. The type of cutting is entered in the central area as being wedge shaped.

98. ABRASION. The figure is entered to the labial of the lower left cuspid, and the blue circle on the tooth chart indicates that the abrasion is confined to the point of the cusp.

NAME		RES.		NPIR		ADDRESS			
NUMBER		TEL.						EXAM. NO.	
BUS.								DATE	
TEL.								NEXT EXAM.	
AGE									

RIGHT		LEFT	
OCCLUSION 123 PATIENT CARE		CARIES 123 123 GINGIVAE	
REFERRED BY REPORT TO			
BI TE WIN QS X - D RA YS NM CA SE HIST ORY MOD ELS PHO TO		PR EV. SER VICE ES TI- MA TE REP ORT PATI ENT ME MO FIL ED INS TR FOR WD	

FIG. 6E. Exhibit of recordings of dental services to be performed. The illustration is about two-thirds the actual size of the card.

MEMORANDA OF TREATMENT.

Memoranda of the various methods that are commonly employed in the treatment of pathological conditions of the teeth and their supporting structures may be entered on the examination card, as a convenient guide for the operator. The several items, and the symbols used, are listed on the chart which accompanies Figure 5. It will suffice to give a few examples which will be illustrated on the examination card in Figure 6E.

In entering memoranda as to treatment on the examination card, it should be understood that the *record of the treatment* is to be made on the patient's permanent record card or chart.

RECORD OF PREVIOUS OPERATIONS. It is desirable to make a record of operations that are in place in a patient's teeth on the occasion of the first examination. These should be indicated by the same symbols that are used for new operations to be performed, but should be entered in red.

In the accompanying examination card, a bridge is recorded which consists of a crown on the upper right second molar, a dummy for the first molar, and a disto-occlusal inlay in the second bicuspid. This is a fixed bridge, the dummy being soldered to both

the crown and inlay. There is a mesio-occlusal restoration in the upper right first bicuspid and a distal gold foil restoration in the cuspid. There is a disto-occlusal restoration in the upper left first bicuspid, which is to be replaced; it was first marked in red, then in black. In the upper left second bicuspid there is a secondary decay at the gingival margin of a disto-occlusal restoration, and a repair is to be made. There is a restoration in the occlusal of the lower left first molar.

PREVENTION OR IMMUNIZATION. The indication for preventive or immunization service should be recorded with the same symbols or marks as for decays, except that they should be in blue in all cases in which the markings might otherwise be confused with the indications for restorative service. For example, when the enamel is only slightly etched in a particular area, and it is decided to immunize the surface with silver nitrate, the decay, whether in a pit, or on a proximal surface or a gingival third position, should be marked in blue. Or, if it should be decided in the case of a recently erupted tooth, with occlusal defects but no decay, to apply copper cement as a temporary procedure, without cavity preparation, the pit should be marked in blue. The use of silver nitrate or copper cement, or whatever other preventive service is performed, is subsequently entered on the patient's permanent record. On the accompanying card, Figure 6E, the buccal surfaces of the lower left first and second molars are marked to be immunized with silver nitrate. The occlusal surface of the second molar is marked for preventive treatment; usually this would be copper cement to carry the case along until a permanent operation might be made, or silver nitrate might be applied to prevent the further progress of a very shallow decay.

The lower left first bicuspid has a distal cavity and the mesial surface of the second bicuspid is marked to be immunized with silver nitrate at the time when the cavity in the first bicuspid is prepared.

RESTORATIONS. Decays usually require restorations and no special markings are necessary, as for example, the occlusal pit and the gingival third decay on the buccal surface of the upper left second molar, and the mesial decay in the first molar, which generally calls for a mesio-occlusal restoration and may be marked as such at the time of the examination. A new disto-occlusal restoration is to replace a previous operation in the upper left first bicuspid, and a repair is to be made at the gingival of a previous disto-occlusal restoration in the second bicuspid. As mentioned above, a disto-occlusal restoration is to be made in the lower left first bicuspid.

A gum resection to eliminate pockets on the proximal and labial surfaces of the lower incisors is indicated by the curved lines

which extend through the interproximal spaces and are connected on the labial surfaces.

A single radiograph is to be made of the upper left lateral incisor which is marked for a porcelain jacket crown. A full set of radiographs, or a set of bite-wing films would be marked in the appropriate square in the lower left corner of the card.

On the lower right, the second bicuspid was marked missing, and it was afterward decided to make a bridge, which is indicated to be made with a mesio-occlusal gold inlay in the first molar, to which a dummy is to be attached with a rest on a disto-occlusal inlay in the first bicuspid.

The methods of recording other types of bridges and partial dentures are indicated on the chart accompanying Figure 5.

Full dentures are indicated by the fact that all teeth of an arch are marked as missing or to be extracted. The type of denture may be entered in the central oval and the condition of the supporting tissues should be indicated on the models.

Alveolar resections may be entered on edentulous mouths in the same manner as gum resections when teeth are present.

Operations in minor oral surgery, which are frequently performed by the general practitioner, may usually be indicated on the examination card. These include gum resections, extractions, root resections and amputations, trimming of the bone for dentures, acute and chronic abscesses, cysts, necrosis, etc. Those that are not listed on the chart may be indicated in the center oval on the card.

Additional entries in connection with any service may be made in the center oval or on the reverse side of the card.

Special Examinations

A special examination is called for in any case of urgency, such as pain, or in relation to any condition out of the ordinary which might be either within or without the strictly dental field; also frequently, for persons who are suffering from some condition that might be secondary to a focus of infection, to determine whether or not there are foci in the mouth. As has been stated, the routine examination should include a careful survey of all of the tissues of the mouth, yet it is recognized that, with few exceptions, the lesions of the soft tissues and those definitely within the field of oral surgery, require special consideration. Therefore, the diagnostic symptoms and signs of all other conditions than those ordinarily covered by the routine examination are arranged in the following groups for consideration and convenient reference:

Pain associated with Oral Lesions.

Foci of infection in the mouth.

Surgical disorders of the mouth, jaws and salivary glands.

Diseases of the soft tissues of the mouth.

Skin diseases; mouth manifestations.

Systemic diseases; mouth manifestations.

In considering the diagnosis of diseases of the pulp and periapical tissues, there were included certain conditions, such as acute and chronic alveolar abscess, root cysts, etc., which more properly belong in the field of oral surgery. This was done for the reason that many general dental practitioners regularly treat such conditions and it seemed desirable to place together the entire chain of diagnostic symptoms, from hyperemia of the pulp to the acute alveolar abscess. However, these conditions are also listed in their proper places in the oral surgery group, with references to the symptoms of each as previously presented.

RECORDING SPECIAL EXAMINATIONS. For many of the conditions for which the diagnostic data are given in the following pages, it will be sufficient to enter the name of the disease or abnormality in the center oval on the examination card. For others, a special case history should be written. The dentist who will take the time to carefully record, in complete detail, the symptoms and signs, with accurate descriptions of lesions, in all cases requiring special examinations, will soon so improve his keenness of observation that he will tremendously promote his ability as a diagnostician. The written description is of much value in this connection as a matter of definitely fixing the symptoms and signs in one's

consciousness for future use. One may test his ability by examining carefully any simple lesion, as for example, a canker sore, and then writing a description of it. Until one has trained himself in doing this, he will find it necessary to reexamine the lesion, possibly several times, to complete the description. If he will then compare his effort with the description in the text, he will probably find that his record is still short one or more important items which are necessary to distinguish this lesion from another.

For many cases, a plain card, the same size as the examination card, may be used for special case histories. This may be filed in its proper place with the other examination cards, or in a special file with a cross reference in the center oval of the regular examination card. Dentists who are engaged in specialty practice generally use printed forms for recording case histories.

It is understood that many diseases which have mouth manifestations are entirely outside the field of the dental practitioner, yet the dentist's opportunity to inspect the mouths of many persons more frequently than does the physician, makes it possible for him to occasionally aid the physician and serve the patient by the early diagnosis of mouth lesions. The dentist is in such large measure responsible for the health of the mouth that he should be familiar with all of its more common diseases.

DIAGNOSTIC INFORMATION AND ILLUSTRATIONS.

Much of the diagnostic data relating to diseases and conditions outside the field of the general dental practitioner has been compiled from a critical review of a limited number of books and writings, and general acknowledgment is made to the several authors. The list of books is given below. Special credit is given in those instances in which the descriptions, as presented, are in part quoted or are closely parallel to the originals. As a general rule, the descriptions given here are less complete in some minor details and no references are made to case histories, many of which are presented by several of the authors.

References are given in this work to the illustrations in the books reviewed, rather than to attempt their reproduction here. The following pages contain references by figure numbers to 538 illustrations of conditions which are outside the field of the present day service of the large majority of dental practitioners. These illustrations are, except in one atlas, accompanied by the descriptions of the diseases and their treatment; thus the figure numbers are also a guide to the related text. Only a few books were selected as reference works for this presentation with the thought that dentists and students, who are particularly interested, would have one or more of them and could secure the others, thus providing themselves with all of the material reviewed, including the illustrations and the recommendations as to treatment.

The pathology and diagnosis of all conditions affecting the teeth and their supporting structures are fully illustrated in the four volumes constituting this work on operative dentistry.

The following books and writings were reviewed in connection with the preparation of this chapter on the diagnosis of oral diseases.

Lectures on Oral Surgery, Thomas L. Gilmer, 1907.

Diseases of the Mouth, F. Zinsser, translated by John Bethune Stein; Rebman Co., New York, 1912.

Atlas der Mundkrankheiten, Morol and Frieboes; Vogel, Leipzig, 1924.

Diseases of the Oral Mucosa, John A. Fordyce; Archives of Dermatology and Syphilology, Volume 13, May, 1926, page 601.

Oral Surgery, Vilray P. Blair and Robert H. Ivy; C. V. Mosby Company, St. Louis, 1928.

Lehrbuch der Mund- und Rachen-Krankheiten, edited by Josef Berberich; George Thieme, Leipzig, 1932.

Clinical Pathology of the Jaws, Kurt H. Thoma; Charles C. Thomas, Springfield, Ill., and Baltimore, Md., 1934.

Diseases of the Mouth and Their Treatment, Herman Prinz, and Sigmund Greenbaum; Lea and Febiger, Philadelphia, 1935.

Clinical Diagnosis of Diseases of the Mouth, Louis V. Hayes; Dental Items of Interest Publishing Company, Brooklyn, 1935.

Laboratory Diagnostic Examinations

A number of diseases require special laboratory examinations as a part of the diagnostic procedure and a brief outline of these is presented. This includes examinations made for the purpose of controlling treatment and of making vaccines. Laboratory diagnosis is helpful in determining the nature and character of an obviously diseased condition. Negative results are less dependable than positive findings, yet reports of positive findings in the absence of clinical symptoms should be viewed with suspicion and are generally of relatively little diagnostic significance. The several examinations are briefly outlined in the following paragraphs.

1. *Stained smears* of sputum and other material from the mouth. Examination should be made of material from around and between the teeth and from sores and ulcers, in cases in which any of the following conditions are suspected: acute ulcerous gingivitis, tuberculosis, diphtheria, gonorrhoea, thrush, actinomycosis, etc., also smears of pus and material from abscesses.

2. *Bacteriologic cultures* of material obtained from mouth lesions and from extracted teeth should be made for cases of suspected diphtheria, for the determination of the type of organism in cases of cellulitis, abscess, etc., for making vaccines and for the testing of the sterility of root canals during treatment.

3. *Dark field and hanging drop examinations* of unpreserved and unstained material for amoeba, spirochetes of syphilis, yeast and other organisms.

4. *Blood examinations.*

Clotting time. Only when profuse bleeding is anticipated or encountered.

Hemaglobin test. When anemia is suspected.

Cell counts. White cells in suspected pyogenic infections and diseases of blood forming organs, as leukemia and agranulocytotic angina. Red cells in suspected anemia.

Stained smears. Differential counts in suspected leukemia, lead poisoning, infection by animal parasites and for determination of Arneth index.

Blood serum. Kahn and Wasserman tests for suspected syphilis, other complement deviation tests.

Blood grouping tests. Preceding blood transfusions.

Chemical examination. Quantitative determination of calcium and phosphorus, especially of calcium in suspected cases of parathyroid disturbance, quantitative sugar determination in suspected diabetes.

5. *Urine examinations.* For sugar in suspected diabetes, for albumin and casts in suspected kidney disease.

6. *Cyst fluid examination.* For cholesterin crystals.

7. *Saliva, chemical examination* for calcium and phosphorus, also for index of susceptibility to dental caries.

8. *Tissue examinations.* All tumors and growths excised should be examined as a general routine, except apical granuloma, gingival material obtained from therapeutic gingivectomy, and that removed in preparing the mouth for dentures. In cases in which malignancy is suspected, a thorough laboratory examination should always be made.

Pain Associated with Oral Lesions

Complaint of pain in the oral region may be due to a lesion within the dental field, to a disease involving the fifth nerve, or to a condition occurring elsewhere, which may cause a reflex pain in the mouth or face. The dentist is also interested in oral lesions which reflexly cause pain elsewhere.

In the most simple case of an inflamed pulp, occurring as the result of a cavity of decay of considerable size, the patient will usually be able to locate the tooth, because it is painful when a particle of food is pressed into the cavity or when the carious dentin is touched with a tooth-pick or the tooth brush. However, if the cavity is in a proximal surface of a tooth and the patient has not discovered its presence, he will be unable to locate the pain in the particular tooth, and he cannot tell whether it originates in the upper or lower jaw, since all of the teeth on either side of the mouth are supplied by branches of the fifth nerve, which in this case carry only painful sensations and not those of touch.

In many cases the pain resulting from a pulpitis may be reflected to some distant part and the patient's attention will not have been attracted to the teeth. When a metal restoration is so close to the pulp as to establish a slight chronic inflammation, the patient may complain of headaches at intervals during several months without suspecting a tooth.

Inflammations involving the supporting structures of the teeth may usually be approximately located by the patient, although, in occasional cases of slight apical pericementitis, there may be confusion in his mind whether the lower or upper tooth is painful to pressure, because his only observation has been of pain when the teeth are brought into occlusion.

PATHWAYS OF PAIN.

The pathways of pain in which the branches of the fifth nerve participate are so complicated as, in some cases at least, to defy analysis. The teeth on either side of the mouth have frequently been compared to sixteen telephones on a single party line. That explains only the patient's difficulty in locating a pulpitis when the cavity is not apparent. If the effort should be made to use the telephone system for further illustration of the pathways of pain in which the fifth nerve participates, one might consider a nucleus in the brain as the local station where all calls from the immediate region are registered. These calls, also those received by other nuclei, representing similar local exchanges, are relayed to a second station, the thalamus, at the base of the brain. This station in turn, and many other secondary stations, pass the impulses on to the super station where bodily sensations become conscious im-

pressions. When one considers that there are twelve cranial nerves, each of which has its own "central exchange," and that the corresponding functional component fibres of a number of nerves are brought together in other "central exchanges," also that there are numerous cross connections between "exchanges," including those to cervical and other nerves, the opportunities for confusion are almost unlimited. As a matter of fact, a complete determination of the sensory distribution of the cranial nerves and their central connections has not been made.

Some idea of the complexity of the situation may be gained from the following brief statement prepared by Dr. Karl L. Vehe:

"The subdivisions of common sensation made by Head and his associates have been followed by a series of experimental and clinical observations on the pathways by which they reach the central nervous system and, in turn, the pathways by which they reach the higher (conscious) centres. The discriminations of touch and temperature of the skin are carried by the fifth nerve and the deep sensibility to pressure and pain is carried by the seventh. The ninth nerve has a fairly definite sensory distribution. Close association with the vagus and the fifth, seventh and ninth central connections may bring to oral structures sensations of pain from pathologic processes as widely separated as the cranium and pelvis.

"This is further complicated, as stated by Head, by the fact 'that where a painful stimulus is applied to a part of low sensibility, in close central connection with a part of much greater sensibility, the pain produced is felt in the part of higher sensibility, rather than in the part of lower sensibility to which the stimulus is actually applied.'

"In obvious lesions there is no difficulty in localizing the cause. In obscure local lesions and in pain of a referred type, some knowledge of the internal relations and connections of the cranial nerves is necessary. This is of even greater significance than the peripheral distribution of the sensory areas, because the cranial nerves are made up of functional components which follow separate routes to their respective destinations within the brain. These functional components are in turn gathered together with similar components from other nerves into a single terminus or nucleus.

"This nucleus is the end station of the neurone of the first order. A single sensory nerve cell may have its peripheral process lying in the skin in relation to a receptor, with the cell body lying in one of the sensory ganglia, such as the Gasserian or Petrosal, and its central process extending into the brain and ending in a nucleus. In the nucleus lie the cell bodies of the neurones of the second order.

"The sensory neurones of the second order carry the impulse from the nucleus to the thalamus at the base of the brain, which is the great sensory station, and sensory impulses from all over the

body arrive here, chiefly by way of the medial lemniscus in the medulla.

“Neurones of the third order take up the impulse from the thalamus and it is carried out through the internal capsule and corona radiata to the sensory area of the brain, where it is interpreted into consciousness.

“When the twelve pairs of cranial nerves are considered there are seven distinct functional components. Of the seven components we are interested in two. First, the general visceral afferent fibers, which carry sensation from the posterior part of the tongue, pharynx, larynx, trachea, esophagus and the thoracic and abdominal viscera. These fibers come into the medulla oblongata through the seventh, ninth and tenth cranial nerves. Being of a common function they are gathered into a single nucleus — the nucleus of the tractus solitarius. Their pathway beyond here is not clear, but since their function gives rise to conscious sensations they must in some way reach the cortex.

“The second group of sensory fibers consists of those of afferent function from the frame-work of the body — the somatic structures. These are chiefly carried by the trigeminal nerve in its wide distribution to head, face and oral cavity. A smaller number of fibers come in over the vagus and glossopharyngeal nerves from the external ear. Here again the fibers of the same function are brought together into the main sensory nucleus and the nucleus of the spinal tract of the trigeminal nerve. Sensations carried into these nuclei are carried to the thalamus by the ventral and dorsal secondary afferent paths of the fifth nerve.”

MANY OPERATIONS INEFFECTIVE IN RELIEVING PAIN.

“Tooth impactions and other obscure dental lesions which may cause distant or referred pain out of the oral region, may lead the dentist to do unnecessary operations for atypical facial neuralgias. More frequently, teeth have been extracted without giving relief, when pain from a distant cause was reflected in the jaws.

“Malignant tumors of the nasopharynx, in close relationship to the auditory tube, gasserian ganglion, sella turcica, or to the second, third, fourth, the second and third divisions of the fifth, the sixth, ninth, tenth, eleventh and twelfth cranial nerves, may produce widespread pain with no local nasopharyngeal symptoms. In 12 of 79 cases reported, teeth had been removed in the hope of relieving the pain in the fifth nerve area.*

“Tonsillectomy and extensive oral operations have been performed for lesions in the region of the cavernous sinus before a diagnosis was made. Mastoid disease, carcinoma of the base of the tongue and larynx, oesophageal stenosis all may cause distress referred to the oral structures.

“The descending root of the fifth nucleus receives also sensory

*New, G. B., Jnl. Amer. Med. Assn., Vol. 19, No. 1, p. 10.

fibres from the seventh, ninth and tenth so that pain may be referred from one area to another. For example, the ear is supplied by the fifth, seventh, eighth, ninth and tenth nerves so that the ear may refer pain to the areas supplied by these nerves or it may receive referred pain from similar areas.

"This information should put the dentist on guard against performing operations if no actual oral lesion exists and should prompt him to insist on a careful analysis of the entire cranio-cervical nervous system."

PROCEDURE IN DIAGNOSIS OF CAUSE OF PAIN.

The first step toward making a diagnosis of the cause of pain should be to secure from the patient as complete a description as possible of the pain itself. Inquiry should be made as to its onset, quality, intensity, duration, etc.

When was the pain first noticed?

Is there a particular point where the pain is first noticed, does it begin as a sudden sharp pain, or is it of gradual onset and does it spread over a considerable area?

How long does it last?

In exactly what region or regions is it felt?

Is it always confined to one side of the head?

How severe is it? The intensity of the pain, as reported, will vary in accordance with the irritation causing it and the reaction of the particular patient. The quality of the pain is therefore a relative term.

Does it prevent the patient from reading or performing his regular duties; or from going to sleep on retiring?

Does it reappear at more or less regular intervals, and is its occurrence associated with any other conditions or circumstances?

Does it result from thermal shock?

Is it more severe when lying down?

Is it related to the use of the teeth in mastication?

These and other questions should enable the dentist to arrive at an accurate conception of the nature of the pain, which should direct his examination. If there is difficulty in locating the cause at once, radiographs should be made to assist in the further study of the case. In many cases, the radiographs will either indicate the cause or suggest lines of inquiry.

In the majority of cases, the pain will be due to a local lesion about the teeth — a hyperemic pulp, pulpitis, gingivitis, pericementitis or a sequella of these conditions. It may be a retained root, the resorption of a root which has exposed a pulp, or any of the conditions discussed in connection with the routine dental examination.

The surgical lesions of the mouth should be considered, in connection with the radiographs. Sequellae of chronic infections including maxillary sinusitis; also impacted, partially erupted and

abnormally developed teeth, supernumeraries and the several benign and malignant tumors of the mouth, including cysts, should be excluded.

In cases of severe pain which suggests the possibility of trifacial neuralgia, it will often be best to consider this affection first. Generally there is little difficulty of making a diagnosis or excluding it from further consideration. In milder cases of neuralgia of the fifth nerve, a very careful analysis will be necessary.

The consideration of trifacial neuralgia will generally exclude or lead to further inquiry regarding glossopharyngeal neuralgia.

It must be remembered that atypical neuralgias will cause the patient to locate pain in the distribution of the fifth nerve, when the cause is remote. It is the duty of the dentist to thoroughly examine the oral cavity and if he finds no apparent cause for the pain, the patient should be referred to other specialists, particularly the aurist, rhinologist, oculist and neurologist, with a report of the examination that he has made.

Hysteria must also be thought of in those cases in which great difficulty is encountered in securing an exact account of the nature of the pain. Positive statements of pain in regions which do not correspond to nerve distribution point more definitely to this condition than vagueness in description, as many persons seem to have difficulty in giving an exact report of the pain.

Diagnostic data relative to the conditions referred to above are given in the following paragraphs.

TRIGEMINAL NEURALGIA. TIC DOULOUREUX. The symptom of this condition which overshadows all else is pain occurring in paroxysms. Blair and Ivy thus classify the symptoms:

1. The neuralgia is confined to the distribution of one or more branches of the fifth nerve.

2. The pain in every case begins in a rather definite spot, from which it may radiate in various directions over areas to which the fifth nerve is distributed and on the same side.

3. The pain is always paroxysmal. The first pain suffered by the patient lasts a short time—from a second to a few minutes, as a rule; it may be slight or severe. The second pain may occur in a day, a month or a year, and as time passes the intervals between pains will be less, and the length and intensity of the individual attacks will be increased. To the paroxysmal character, there may be later added an irregular periodicity.

4. The pain always returns in the same spot; it may radiate, but will always be sharpest in that point. Other pain areas may develop and they in turn are equally definite. After some months or years the primary area may be less severe. The second and third divisions are the ones most commonly affected.

5. The neuralgia is usually confined to one side, but involvement of both sides is not uncommon.

6. In most cases there are spots over the distribution of the nerve, particularly at the exit foramina, stimulation of which by pressure causes a twinge in the pain area.

7. The touch of the finger, a breath of cold air, in some cases heat, taking food into the mouth, the act of swallowing, or a sudden movement may bring on the pain.

8. In most cases the first pain appears after the forty-fifth year, one case occurred at nineteen.

"The observation of a patient in the midst of an attack of major trigeminal neuralgia when once seen is never forgotten. A patient affected with this disease who has been conversing naturally, suddenly puts his hand to his face, screws up the side of the face, the nose runs and the eye on the affected side may shed tears. He appears to be in extreme pain. 'The essential feature is the spasm or paroxysmal character of the pain. Like a flash of lighting it comes and goes and between the paroxysms the patient is pain free,' and is again conversing with the observer." (Prinz and Greenbaum.)

Illustration: Prinz and Greenbaum, Figure 254.

GLOSSOPHARYNGEAL NEURALGIA occurs along the distribution of the glossopharyngeal nerve. It is a rare disease, but the pain is very severe. Usually it starts suddenly in the throat at the base of the tongue and in the region of the tonsils, radiating to the front of the ear and sometimes down the neck. The pain may be induced by swallowing. (After Prinz and Greenbaum.)

ATYPICAL NEURALGIA. Neuralgic pains of the face, head and neck, other than those of trigeminal neuralgia, may result from infections or trauma. They are most prevalent in middle life. The cause of pain occurring in and about the teeth may be located anywhere between the origin of the nerve in the brain and its end branches, particularly the teeth and their immediate surroundings. Any inflammation or irritation about the teeth or within the jaw bone may cause such neuralgia. Similar neuralgias in the oral region may be reflected from irritations almost anywhere between the top of the head and the pelvis.

HYSTERIA is a mental disorder characterized by the appearance of peculiar physical symptoms which may assume various forms, reaching into the sphere of sensation and mobility, but do not correspond to anatomical nerve distribution. Such symptoms occur in individuals with inadequate personalities, who find it difficult to adjust themselves to their environment. (Prinz and Greenbaum.)

PHOBIAS are unexplainable and uncontrollable fears of some serious condition such as cancer, which develop in relation to pain or swelling in any part of the body; frequently the mouth and especially the tongue. Such persons are overly apprehensive of physical symptoms. (Prinz and Greenbaum.)

Foci of Infection in the Mouth

There are two principal types of chronic foci of infection in the mouth; the chronic apical abscess or granuloma and chronic suppurative pericementitis or the pyorrhea pocket. The diagnostic data for both conditions have been given under the appropriate headings. Of less frequency are chronic infections of inflamed gums caused by partially erupted teeth, jagged edges of teeth or projecting margins of restorations. Infection may also be caused via the blood stream or lymphatics directly from an inflamed pulp without noticeable involvement of the periapical tissues, as in cases of infection of the cervical glands by tubercle bacilli and other organisms which gain access to the pulp through open cavities of decay. In some cases pain, often headache, is the principal symptom of a chronic pulpitis which may continue for months before the pulp eventually dies. Acute periapical abscess, and less often acute infections of deep pyorrhea pockets, may cause concurrent acute infection elsewhere, as a myositis, neuritis or arthritis.

In view of the fact that many serious secondary lesions occur as a result of such foci, also because at least seventy-five percent of adults have one or more chronic mouth foci, it seems desirable to set up a separate plan of examination, together with a form of report to be sent to physicians who refer cases to the dentist for examination.

A full set of radiographs should be made previous to the examination. However, the point should be emphasized that the diagnosis should never be made from radiographs alone. Physicians should not undertake to make a diagnosis, as they would necessarily depend solely upon radiographic examination. It is admitted that radiographic evidence might be such as to warrant the extraction of an occasional abscessed tooth, but there is seldom a case in which other findings are not necessary to secure all of the available evidence. For example, a case may be cited in which the radiograph showed a large cavity in the bone into which the roots of both upper bicuspids apparently projected, and on the basis of a radiographic examination alone, both teeth should have been extracted. The dentist tested both teeth for pulp vitality; the first bicuspid responded normally, the second not at all. Therefore only the second bicuspid was removed. The rarefied area was evidently entirely to the lingual of the first bicuspid root, as the bone healed promptly following the extraction and the pulp of the first bicuspid was vital when tested several years later.

Excellent radiographs, which show the structure of the bone about all of the teeth very clearly, are necessary to a proper diagnosis. One should have opportunity to compare regions of possible rarefaction with definitely normal bone. The various anatomical landmarks should also be clearly discernible.

The involvement of the peridental membrane and alveolar process by chronic suppurative pericementitis should be determined

by the methods suggested in discussing the diagnosis of that disease. Particular attention is again called to the fact that instrumental examination to determine the depth of pockets is generally more reliable than radiographs, particularly for the lingual and buccal-labial sides of roots. It is desirable however, to use radiographs in all cases.

The condition of pulps should be determined in all cases in which there is any question as to their vitality, and the periapical tissues should be examined in the radiograph, as directed in the diagnosis of chronic periapical infection.

A critical visual and instrumental examination should also be made of the mouth, together with the radiographs, to find possible areas of inflammation about impacted or partially erupted teeth, buried roots, areas of residual infection following the removal of teeth in cases of periapical cysts which were not completely enucleated, etc.

REPORT TO PHYSICIAN. The report to a physician in all such cases should be so full and complete as to be in itself convincing as to its thoroughness. It should contain the following information.

The appearance of the mouth as to health and cleanliness.

The dentition, which may be a report of the teeth missing, or of those present, whichever would make the shorter list.

The condition of the gingivae and peridental membranes in as much detail as may be necessary; dividing the teeth into three groups or less, as conditions may warrant: (1) Teeth with supporting structures in good condition; (2) Areas of gingivitis or shallow pockets, which can be satisfactorily cared for by local treatment; (3) Deep pockets; with depths in millimeters, and additional information regarding each tooth as to mobility, etc., with recommendations as to extraction, or the surgical elimination of pockets.

The condition of the teeth as to pulp vitality, in four groups, or less, as may be desirable: (1) Teeth having vital pulps; (2) Teeth with inflamed or dead pulps or poor root fillings, but no involvement of the periapical tissues, which should have prompt treatment; (3) Those having good root canal fillings and no involvement of periapical tissues, which are considered free from infection; (4) Those which show rarified apical areas, with recommendations that the teeth be extracted, or in some cases that roots should be resected or amputated.

In cases in which the person referred by the physician is the patient of another dentist, a copy of the report should, as a matter of courtesy, be sent to the dentist. As a general rule, it will be best for the dentist who makes the examination, not to discuss his findings with the patient.

Surgical Disorders of the Mouth, Jaws and Salivary Glands

ABNORMALITIES.

CLEFT PALATE AND HARELIP. These are congenital defects which may involve the hard and soft palate, the alveolar process and lips. In a double cleft palate and harelip, the defect is an opening connecting the mouth and nasal cavity in the form of a letter Y, the stem of which represents a failure of union of the palatal processes of the maxillary bones and the horizontal plates of the palate bones along the midline of the palate, while the two short arms represent the failure of union of the palatal processes with the fronto-nasal process. There may be a failure of union of any portion of the stem only of the Y, or of either side of the fronto-nasal part, or of the stem and one side of the fronto-nasal part together, or of either side of the fronto-nasal part alone, or of one or both sides of the lip alone. There very rarely occurs a cleft through the median line of the palate and of the alveolar process, gum and lip between the central incisors, or a median cleft of the lip alone.

The molars and generally the cuspid teeth are in the maxillary portion and the central incisors in the frontal portion. The lateral incisors are usually missing, but are likely to be in the frontal portion if they are present.

Illustrations: Blair and Ivy, 172 to 194; Prinz and Greenbaum, 212, 213, 214.

TORUS PALATINUS. An elevation of the bony palate in the median line, which occurs frequently in adults. It gives neither pain nor discomfort.

Illustrations: Prinz and Greenbaum, Figure 216; Hayes, Figures 97, 98.

ABNORMAL SUPERIOR LABIAL FRENUM. This frenum is often unusually large and may extend between the central incisors, with some fibres attached to the alveolar process on the lingual side of the arch. It may contribute to the maintenance of a diastema between these teeth.

Illustrations: Hayes 55; Prinz and Greenbaum, 221, 222.

ABNORMAL LINGUAL FRENUM. TONGUE TIE. The frenum connecting the lower surface of the tongue to the floor of the mouth may be very short and may be attached to the alveolar process,

thereby limiting the movements of the tongue, causing a peculiar defect in speech.

Illustration: Hayes, Figure 69.

UNERUPTED TEETH. See The Dentition.

IMPACTED TEETH. See The Dentition.

SUPERNUMERARY TEETH. See The Dentition.

RETRUSION OF THE MANDIBLE. An underdevelopment of the mandible which produces a receding chin. This may result from any interference with normal growth, such as an ankylosis of the temporo-maxillary joint.

Illustrations: Blair and Ivy, Figure 235. Thoma, Figure 6.

HYPERTROPHIES.

PROGNATHISM. An overdevelopment of the mandible which causes a protrusion of the chin.

Illustrations: Hayes, Figure 159; Blair and Ivy, 233, 234.

HYPERTROPHIC GINGIVITIS. A chronic inflammation with proliferation of the gum tissue by an increase in the size and number of the cellular elements. The gums may in some cases almost completely envelop the crowns of the teeth.

Illustration: Hayes, Figure 10.

MACROCHEILIA. LYMPHANGIECTASIS. A hyperplasia of either lip, and occasionally of both lips, involving principally the lymph vessels. The blood vessels may also be abnormally developed. The lip may be two or three times its normal size and so everted as to cause a very objectionable deformity. The condition is painless.

Illustrations: Hayes, Figures 43, 45; Prinz and Greenbaum, Figure 219; Fordyce, Figure 2.

ACQUIRED BLOOD VESSEL VARICES, which are not tumors, are frequently observed on the under side of the tongue. They are varicose veins. Lymphatic varices are occasionally seen on the lip, tongue and buccal mucosa. They are white, clear pseudo-vesicles. They disappear on pressure, and when punctured, they ooze lymph for a considerable time.

DENTAL INFECTIONS AND SEQUELLAE.

TRAUMATIC APICAL PERICEMENTITIS. See Diseases of Dental Pulp and Periapical Tissues.

ACUTE APICAL PERICEMENTITIS. See Diseases of Dental Pulp and Periapical Tissues.

CHRONIC APICAL PERICEMENTITIS. PROLIFERATIVE PERICEMENTITIS. GRANULOMA. ABSCESS. See Diseases of Dental Pulp and Periapical Tissues.

PERIAPICAL CYST. RADICULAR CYST. ROOT CYST. See Diseases of Dental Pulp and Periapical Tissues.

ACUTE APICAL ABSCESS. ACUTE ALVEOLAR ABSCESS. See Diseases of Dental Pulp and Periapical Tissues.

CONDENSING OSTEITIS. OSTEOSCLEROSIS. The solidification of bone of the jaws associated with infection or injury is common. As a rule there is no change in the contour of the bone and generally there are no symptoms. The radiograph reveals an opaque area corresponding to the sclerotic bone.

Illustrations: Thoma, Figures 63, 67, 300a, 300b.

PERIOSTITIS. An inflammation of the periosteum may result from injury, infection, or chemical or metallic poisoning. Practically all operations involving the maxillary bones, including the extraction of teeth, cause a periostitis of greater or lesser degree. A suppurative periostitis may occur in cases of traumatic infection or acute periapical (alveolar) abscess. When the pus reaches the periosteum, it may strip the periosteum from the bone and form a sub-periosteal abscess between the bone and the periosteum. The local symptoms of periostitis are pain, swelling and tenderness to pressure. In cases of a sub-periosteal abscess the pain is exceptionally severe and the swelling is broad and flat. Both will continue until drainage is established. The systemic symptoms will vary according to the character of the injury or infection.

ACUTE OSTEITIS. An acute osteitis within the maxillary bones usually results from a periapical abscess, from a peridental infection or from an infection occurring about the crown of a partially erupted tooth. It may also result from trauma, or from chemical or metallic poisoning, plus infection. This condition is an intermediate step in the formation of all bone abscesses. Both the local and systemic symptoms depend upon the character of the injury and the type of infection. The region will be painful with usually little swelling, while the inflammation is confined within the bone. The area will usually be tender to pressure at all times.

Illustration: Thoma, Figure 60.

CHRONIC OSTEITIS. This term is applied to a slowly progressive, infectious destruction of bone, which has often been referred to as *caries of bone*. It is a very mild suppurative process, usually without pain or other symptoms, and may destroy considerable bone cell by cell over a period of years. It occurs more frequently in the upper jaw than the lower. There may be one or more sinuses from which pus, usually of straw color, is discharged. This bone bleeds very little when it is cut away. The teeth gradually become loose as more and more bone is destroyed about them. New bone is not built to replace that destroyed.

OSTEOMYELITIS. An osteomyelitis is an infection of the medullary portion of the bone, which may occur as a result of trauma with infection, or from infection alone, as in case of an acute periapical (alveolar) abscess. It may penetrate all of the

marrow spaces and Haversian canals and is often the intermediate step between an acute infection and necrosis.

Illustration: Thoma, Figures 73 to 80; Blair and Ivy, Figure 127.

NECROSIS. Necrosis of the maxillae and mandible results from infection which involves the periosteum or the medullary portion of the bone. It may result from an acute abscess or an injury which cuts off the blood supply of a portion of the bone, or from mercury, phosphorus or arsenical poisoning. In any pyogenic infection involving the bone, the circulation may be cut off from a region, resulting in its death. The symptoms are those of the condition which causes the necrosis, plus the finding of the necrosed bone. The discharge of pus is persistent, so long as any dead bone remains, and always has a very foul odor. As a rule, three or four weeks are required for the sequestration of a piece of necrotic bone of the average size occurring in the mouth. Small sequestra may be separated in less time. The teeth are often very loose. A sharp steel probe, passed through a sinus or other opening, and placed in contact with the necrotic bone will indicate that it is hard, rough bone, and often honeycombed. The diagnosis should be made without difficulty. Sooner or later, a sequestrum will be formed, which will be movable. The pain is usually severe until there is free evacuation of the pus. In cases of subperiosteal abscess, the raised periosteum may build an involucrum of new bone — usually a thin shell, which will partially envelop the sequestrum.

In the extraction of teeth, portions of the alveolar process may be injured or fractured and become necrosed. They delay the healing of the socket until all of the dead bone is thrown off. This may require from ten days to two weeks, or longer.

Illustration: See Volume IV, Necrosis; Blair and Ivy, Figure 128.

PHOSPHORUS NECROSIS. Persons who handle phosphorus or inhale its fumes are subject to phosphorus poisoning, which may result in necrosis of the jaw bones. The phosphorus formerly used in making matches was particularly poisonous. The condition seldom occurs in mouths that receive proper attention. Particles of phosphorus dust gain entrance to the bone through carious teeth and cause necrosis. The gums are usually inflamed; they become spongy, bleed easily and ulcerate. The breath is fetid and has the odor of garlic. The disease is essentially chronic and consists of an osteitis caused by the phosphorus, followed by infection which results in necrosis.

Illustration: Prinz and Greenbaum, Figure 86.

ARSENICAL NECROSIS was of rather frequent occurrence when arsenic was used for pulp devitalization, due to failure to properly seal the cavity or the application of arsenic to pulps of teeth with roots that were only partially formed. Cases are rarely seen today,

as there is no other use for arsenic which endangers the dental structures.

ACUTE MAXILLARY SINUSITIS. The bony septum between the maxillary sinus and the apices of the roots of the upper molar and bicuspid teeth, and occasionally the apex of the cuspid, may vary in thickness from that of a sheet of paper to possibly three-eighths of an inch or more, and infections from the pulp may easily penetrate a fairly thin bony septum and involve the lining membrane of the sinus. Acute infections of the maxillary sinus are characterized by the usual symptoms of infection, which are increased if pus is confined within the sinus. The pain may be severe and the face may be tender to pressure over the region of the sinus. There is usually headache also. The upper teeth on the affected side should be examined for both apical and peridental infections, which might have penetrated the sinus. An anterior-posterior head radiograph may show a cloudiness on the affected side. A transillumination test may be made in a dark room, by placing an electric lamp in the mouth. With the patient's lips closed, the transmitted light, through a normal sinus, shows a brighter area below the eye than is the case on the affected side, as the fluid in the antrum obstructs the light. There may be a discharge of pus into the nose.

Illustrations: Hayes, Figure 198, 199; Thoma, Figure 98.

CHRONIC MAXILLARY SINUSITIS. The only difference in the diagnosis of chronic as compared with acute infection of the sinus is the mild nature of both the constitutional and local symptoms. There may be only occasional pain or discomfort when the osteum maxillare becomes closed. Pus may discharge into the nose.

ACUTE CELLULITIS. LUDWIG'S ANGINA. This is an acute infection of the connective tissue of the floor of the mouth, with rapidly spreading cellulitis. There may be extreme toxic symptoms, which, combined with embarrassment of respiration, may endanger the life of the patient. There is a hard board-like swelling beneath the border of the mandible on one or both sides. It may force the tongue upward and backward, with impairment of both swallowing and breathing. The infection may come from an apical abscess or any infection which may involve the tissues of the floor of the mouth. The patient will have the general symptoms of a severe, acute infection.

Illustrations: Blair and Ivy, Figures 135, 136; Prinz and Greenbaum, Figures 245, 246.

ACTINOMYCOSIS ("lumpy jaw") is an infectious disease of the ox, swine, horse, and occasionally of man, induced by the micro-organism classed as actinomyces. This disease is seen most often in agricultural areas, and in many cases the infection occurs in the mouth as a result of chewing the stalks of straw of various grains or grasses. It may occur in the chest, abdomen, or elsewhere.

When the skin or mucous membrane is involved, the first symptom is a small, deep seated nodule, generally painless, which enlarges and, as the surface breaks down, there occurs a serous or purulent discharge, that may contain characteristic "sulphur" granules, in which the ray fungus may be observed under the microscope. This ulcer becomes incrustated or heals with a scar, while new nodules appear in the adjacent tissue and the process continues. There is an extensive surrounding area of induration. When the tongue is infected, there is considerable swelling, which may involve the floor of the mouth.

The surface of skin or mucous membrane becomes irregular in folds or lumps, and this, together with deep induration, suggested the name "lumpy jaw." (After Gilmer, and Prinz and Greenbaum).

Illustrations: Prinz and Greenbaum, tongue, Figure 166; cheek, Figure 167; Hayes, face, Figure 174; Plate No. 9, colored; Thoma, microscopic section, Figure 83; Figure 85, colored.

SALIVARY GLANDS AND DUCTS.

SALIVARY FISTULA. A fistula is an abnormal opening through which a normal body fluid is discharged. A salivary fistula may be an abnormal opening from a salivary gland into the mouth or through the skin, or it may be an opening from the mouth cavity through the skin to the surface.

SALIVARY CALCULI. Calculi may form within the salivary glands or ducts. They occur much more frequently in the ducts, particularly Wharton's duct, and are often symptomless. They seldom cause obstruction of the ducts; however, when Wharton's duct is obstructed, the gland will be swollen and painful, particularly at meal time. The stone may be palpated with one finger inside the mouth and one below the chin. Usually the diagnosis may be made with the radiograph.

Illustrations: Prinz and Greenbaum, Figures 231 to 236; Hayes, Figures 135 to 141; Blair and Ivy, Figure 293.

PTYALISM. This is an excessive flow of saliva, which often occurs during dentition, in cases of chronic suppurative pericementitis and in other inflammations of the mouth, also in a number of diseases of the central nervous system.

Illustration: Prinz and Greenbaum, Figure 237.

APTALISM. XEROSTOMA. The lack of normal salivary flow causes a dryness of the mouth and is of significance in connection with the occurrence of dental caries. The more limited the flow of saliva, the more rapid is the destruction of the teeth by decay. Fortunately, cases of extreme aptyalism are very rare.

Illustrations: Prinz and Greenbaum, Figures 238 to 242.

TEMPORO-MANDIBULAR JOINT.

DISLOCATION OF THE MANDIBLE. Forward dislocation of the mandible is rare; it occurs more frequently in females than in males. The condyles slip forward of the eminentia articularis and the mandible is held there by the tension of the muscles. The mouth is wide open and the patient can not move the mandible. The condyle may be felt in advance of its natural position, and may be shown with the radiograph. If the dislocation is unilateral, the chin will deviate to the opposite side.

Illustrations: Blair and Ivy, Figures 87, 88, 89.

SUBLUXATION OF THE MANDIBLE. A partial dislocation of the mandible, usually on one side, often occurs, due to the laxness of the ligaments about the joint, when the mouth is opened very wide, as in gaping or during dental operations. The condyle will move a little too far forward and may prevent the jaw from being closed without assistance.

TRISMUS. Reflex irritation, or an inflammation in the region of the muscles of mastication, may cause a contraction or tenseness of the muscles which prevents or limits the movement of the mandible in efforts to open the mouth. If there is inflammation, the effort may be painful, otherwise there is no pain.

ARTHRITIS. Inflammation of the temporo-mandibular joint, with pain and limitation of movement, may be due to suppurative disease which involves the joint; it may be due to arthritis following scarlet fever, small pox, measles and other acute infectious diseases, or to a gonorrheal infection. An ankylosis of the joint may result.

Illustrations: Thoma, Figures 103, 104.

FALSE ANKYLOSIS of the temporo-mandibular joint may be due to an injury, without involvement of the bones, in the healing of which considerable scar tissue was formed, thus limiting the movements of the mandible.

TRUE ANKYLOSIS of the temporo-mandibular joint. The movement of the lower jaw is definitely limited, although absolute immobility is rare. This condition may be due to a fibrous union of the structures composing the joint, or to actual bony union. The ankylosis may result from any type of arthritis. If the ankylosis occurs during childhood, the mandible is retarded in its development and the chin is less prominent than normal. The teeth are liable to be lost from extensive caries, due to the inability of the patient to clean the mouth, and the inaccessibility of the teeth for care by the dentist.

Illustrations: Blair and Ivy, Figures 279 to 283; Hayes, Figures 153, 154; Thoma, Figure 100.

JOINT STRUCTURE INJURIES AND IMPAIRED HEARING.* Edentulous jaws, or jaws with teeth much reduced in length by abrasion, result in an excessive movement of the mandible in closing the mouth, that may cause distortion of the temporo-mandibular joint with impaired hearing for low-tones; stuffiness of the ears, especially after eating; low buzzing tinnitus; pain within and around the ear; dizziness, slight or prostrating, and often relieved by auditory tube inflation; headache (vertex and occiput), burning in nose, throat and tongue. There may be intervals of improvement in hearing and any of the other symptoms may be present from time to time in a given case. These symptoms may confuse the diagnosis and suggest primary ear disease or oral pathology.

There are two mechanisms which are supposed to cause these symptoms: (1) Pressure on the external auditory canal, especially the cartilagenous part, and trauma to the tympanic plate and deeper ear structures by the loose joint and concussion of the condyle head; (2) the marked overbite, with the relaxation of the temporo-mandibular joint and upper head of the external pterygoid muscle, makes possible the crushing of this latter structure and the meniscus against the cartilagenous and membraneous part of the auditory (eustachian) tube and the tensor and levator palati muscles. This in turn leads to tubal obstruction and middle ear catarrh with all of their sequelae. In addition the auriculo-temporal and chorda tympani nerves may be compressed.

FRACTURES OF THE MANDIBLE AND MAXILLAE.

It is of especial importance that the dentist should be qualified to diagnose and treat fractures of the jaws, in order that the occlusion of the teeth may have proper consideration in the treatment. The large majority of fractures of the jaws are traumatic; a few are pathological. Most of the diagnostic data follow the writings of Thomas L. Gilmer and Blair and Ivy.

Traumatic fractures. In making a diagnosis in cases of traumatic fracture, one should first obtain a history of the accident or blow which caused the injury, also, if possible, the direction of the force and the point of contact. Each fracture should be classified in accordance with the following definitions:

A closed or simple fracture is one in which there is no communication between the outer surface and the site of injury to the bone.

An open or compound fracture is one in which there is an external opening communicating with the bone, thus permitting infection to enter.

A complete fracture is one in which there is a loss in the continuity of the bone as a whole.

*Courtesy of Karl L. Vehe.

A *partial fracture* is one in which a portion of a bone is broken away, without complete loss in the continuity of the bone as a whole.

A *greenstick fracture* is an incomplete fracture with some bending of partially calcified bone, occurring in children.

Fractures may be classified as *single* or *multiple*, indicating a single or several fractures of the same bone.

A *comminuted fracture* is one in which the bone is shattered into several fragments.

An *impacted fracture* is one in which one fragment is driven into the other and fixed there by the force of the blow.

The symptoms of fractures of bones are *pain*, which is aggravated by movement of either fragment; *disability*, which varies according to the use of the bone involved; *swelling*, *tenderness*, *deformity*, *abnormal mobility* and *crepitus*. In cases of jaw fractures, there usually occurs a disturbance of the occlusion, which deserves special consideration.

FRACTURES OF THE MANDIBLE may occur at any point between the condyles, including the coronoid process in its protected position, but are more common in the body of the bone between the symphysis and the angle. The majority of single fractures occur near the position of the mental foramen—in the bicuspid region. Fractures in the incisor-cuspid portion of the bone are less frequent than in the molar region. They seldom occur exactly at the symphysis, but nearly always at least a little to one side, between the central and lateral incisors, or directly under the lateral or cuspid. Fractures may occur anywhere in the molar region and are not infrequent at the angle, posterior to the last molar.

Displacement results in part from the force which caused the accident, but for the most part from muscular contraction influenced by the position and direction of the fracture. A fracture near the symphysis may show no displacement, because the muscular traction is evenly balanced. A diagonal fracture may prevent displacement in the direction of muscular traction.

Disturbance of the occlusion occurs in most fractures of the mandible. This will be observed when the patient attempts to close the teeth.

Abnormal mobility of the fragments is usually detected without difficulty, and *crepitus* may be elicited if the ends of the fragments are in contact.

The *pain*, *swelling* and other signs of inflammation are likely to be more pronounced several days after the injury, particularly if the case has received no attention. In open fractures, the symptoms are often greatly aggravated by infection, which may cause necrosis.

Radiographic examination should generally be made; in occasional cases of closed single fractures, either in the body or ramus, to make the diagnosis; in other cases to verify the diagnosis and discover more exactly the line of fracture, with particular reference to the exposure of roots of teeth; also to survey the bone with a series of exposures to discover other possible fractures, partial or complete.

Illustrations: Muscular tractions and displacements, Gilmer, Figures 7 to 20; Fractures and displacements, Blair and Ivy, Figures 31 to 47; Radiographs of fractures, Hayes, Figures 277, 278, 279, 282, 283, 286; Thoma, Figures 47 to 58.

FRACTURES OF THE MAXILLAE. Fractures of the maxillae are infrequent, as compared with those of the mandible. The majority are partial fractures, in which several teeth, most often incisors are forced upward, thus breaking away the labial plate of bone, or cases in which several teeth, with the entire surrounding alveolar process may be broken away from the remainder of the bone, or more frequently cases in which a portion of the alveolar process is broken away in extracting a tooth. Occasional cases occur in which one or both of the superior maxillae are broken loose from all of their bony attachments, with or without disturbance of the teeth in their respective sockets. By grasping any upper tooth, the entire maxillae may be moved. Radiographic examination is also desirable in fractures of the maxillae.

Pathological fractures. These fractures may result from local or systematic disease which severs the bone by destroying it, or so reduces the bone that it is broken by very slight violence. The presence of neoplasms, cysts and infections are the local causes, while Paget's disease and other systemic conditions involving the bone are general causes of fractures. The damage which these conditions have caused to the bone, are determined by the radiograph.

WOUNDS AND FOREIGN BODIES.

For the diagnosis and discussion of gunshot and other wounds of the face and jaws, including the presence of foreign bodies, one should consult the several excellent text books on Oral Surgery.

PARALYSES OF THE FACE.

FACIAL PARALYSIS. BELL'S PALSY. This is a paralysis due to disease or injury of the facial nerve. It is often caused by exposure to a draft of cool air. This nerve transmits motor impulses to one-half of the scalp and face, except the muscle that elevates the eyelid, the ocular muscles, the muscles of the tongue and the muscles of mastication. It also supplies impulses to one of the muscles of the middle ear, to the stylohyoid and the posterior

belly of the digastric. The chorda tympani, which carries the sense of taste, is for a part of its course incorporated with the seventh nerve.

The symptoms vary with the location and extent of the lesion. A lesion situated in the face area of the cortex, the nucleus of the facial nerve, or the conducting path between, will cause a paralysis of the opposite side of the face. Shortly after the fibres leave the nucleus, they cross the median plane; lesions below this point will cause paralysis of the face on the same side. If the lesion is between the pars intermedia and the giving off of the chorda tympani, there will probably be loss of taste on one half of the tongue. In a complete one-sided paralysis, there will be a smoothing out of the natural creases in the skin of the forehead, with inability to raise or wrinkle the brow, and a slight drop of the eyebrow on that side, also inability to close the eye. When an attempt is made to close the eye, the globe turns upward and there is a slight movement of the lower lid, the latter probably being due to certain muscle fibres innervated through the sympathetic. In a paralysis of long standing, there may be considerable irritation of the eye, due to inability to close it. The buccinator muscle will remain flaccid and food will collect in the buccal pouch on that side. The mouth will be drawn to the opposite side by the unopposed action of the opposite buccinator. It is impossible to pucker the mouth. (After Blair and Ivy.)

Illustration, Blair and Ivy, 303; Prinz and Greenbaum, Figures 247, 248, 249; Hayes, Figures, 165, 166.

FACIAL TIC. This is a motor disturbance affecting chiefly the muscles supplied by the seventh nerve, and characterized by a set of spasmodic contractions of certain muscles, recurring at more or less regular intervals. There are one or more sharp contractions of the affected muscle, followed by a period of relaxation. The interval between spasms varies. It most commonly appears in the zygomatic and orbicularis palpebrarum muscles of one side, but may spread to other groups, and the tongue, neck, shoulder and arm muscles may all become involved. It is essentially a habit spasm. (Blair and Ivy.)

TUMORS. NEOPLASMS.

The classification of tumors, as here presented, is on the basis of the histologic structure and the diagnostic signs and nomenclature follow rather closely the descriptions of Prinz and Greenbaum.

Blair and Ivy state that "nearly all the tumors and cysts which can arise in any part of the body may be found in the mouth, except those which are peculiar to certain extraneous organs. There are also certain tumors and cysts which are peculiar to the mouth."

NEVI. MOLES. BIRTHMARKS. Nevi are circumscribed, pigmented or non-pigmented, flat or elevated malformations of em-

bryonal origin, but may appear at any age. They are of common occurrence on the skin and are less frequent on the vermillion lips and in the mouth. They are commonly congenital. The pigmented nevi consist of variously sized and shaped, more or less round, brown or blackish spots. On the lips, they are pin-head to pea sized, dark slate, blue or black, non-elevated, irregular or rounded spots, usually located on the lower lip. On pressure, the spot not only fails to disappear, but is apt to become more distinct. No infiltration or subjective symptoms are manifest. Occasionally, these degenerate into cancer. If there is a definite increase in size, with induration, this is an indication of danger. (After Prinz and Greenbaum.) Illustration: Prinz and Greenbaum, Figure 260.

EPITHELIAL TISSUE TUMORS.

PAPILLOMATA. This term is often used to designate all sorts of epithelial elevations, including the common wart, which is caused by infection. Papillomata that may be classified as tumors are restricted to epithelial growths resulting from mechanical trauma, chemical and other forms of irritation. Both infectious and traumatic papillomata occur in the mouth; both are painless, smooth, firm or soft, pin-head to split-pea sized, hemispherical elevations with a smooth surface and with or without a pedunculated base. Those which develop as a result of a definite point of irritation, as a jagged tooth, should be observed with suspicion in regard to possible malignancy. A papilloma may develop on any part of the oral mucosa; they occur most frequently on the tongue, and are usually single.

Infectious papilloma of the lip may be located on the mucous or muco-cutaneous surface. It fits the description given above, except that it is softer than the traumatic type, and its color may be gray or vary little from that of the mucous membrane. Traumatic papilloma results from persistent biting of the lip in one spot, the prolonged contact of a jagged tooth, or the irritation of a pipe stem. The lesion is usually firm and markedly fibrous. It may undergo malignant degeneration, and the change may be difficult to diagnose. (After Prinz and Greenbaum.)

Illustrations: Thoma, Figure 336; Hayes, Figures 101 to 105, 201 to 208; Fordyce, Figure 10.

ADENOMATA. These are of glandular structure, firm, nodular, slow growing, completely encapsulated and therefore freely movable. They occur as sebaceous gland adenomata of the oral mucosa. Cysts of the mucous glands are also benign adenomata. These are described with other cysts.

Illustration: Prinz and Greenbaum, Figure 261.

FORDYCE'S DISEASE is a term applied to a condition of the mucous membrane of the lips and cheeks, in which there are small,

light-yellowish, discrete, glandular bodies under the mucous membrane. Fördyce thought this to be a granular change in the protoplasm of the cells. Other writers believe that it results from the blocking of the minute sebaceous glands, which are abnormally placed in the mucosa.

Illustrations: Hayes, Figure 53; Prinz and Greenbaum, Figure 230.

CARCINOMATA. A malignant epithelial growth, which may involve the oral mucous membrane, and very seldom appears before the age of forty. Tissues which are imperfectly formed, as nevi, or those which have been pathologically damaged, as leukoplakia, coupled with chronic mechanical, chemical or biochemical irritation, present the best etiological explanation of these lesions. The following mouth lesions may be listed as possibly precancerous: pigmented nevi; chronic traumatic papillomata; keratoses—any horny or warty growth, particularly on the lips of elderly persons, who are exposed to much inclement weather; cicatrices and old burns; radiodermatitis, especially the keratotic type; erythroplakia (see Lesions of Soft Tissues of the Mouth); lupus vulgaris (see Mouth Manifestations of Systemic Disease, under Tuberculosis), and lupus erythematosus (see Mouth Manifestations of Skin Diseases). Local irritation of any of these lesions may cause the development of malignancy.

Illustrations: Thoma, precancerous lesions, Figures 366 to 374; carcinoma, 377 to 381.

Carcinomata or epitheliomata of lip. It has been estimated that from two to three per cent of all malignant growths are lip cancers. Constant or often repeated irritation, is the most important etiological factor, particularly pipe and cigarette smoking (if the cigarette remains in contact with the lip until nearly consumed). Ninety to ninety-five per cent of labial cancers are in males and about ninety-five per cent of them involve the lower lip. In the list of cancers of the lip in clay pipe smokers, thirty per cent are colored women. The lesion on the lip develops in three ways:

1. It most frequently begins as a keratotic or roughened spot which, after a variable time, begins to scale. Eventually an erosion develops and the area of scaling becomes slightly larger, with a superficially eroded surface and possibly slight crusting. If the crust is removed, the eroded area bleeds. Slight induration gradually develops, and the area enlarges over a period of several months. There is no glandular involvement at this time.

2. In a persistent fissure, usually located in the center of the lip, the first change is the development of a circumscribed induration at its base.

3. As a small, intra-epidermic, smooth, circumscribed induration or nodule, the surface of which erodes early.

When the epithelioma is once established, the base of the lesion is characterized by its induration and its hard rolled, elevated appearing border. There are two types; the vegetative and the infiltrative. In the vegetative type, a cauliflower-like growth of varying size develops, which bleeds easily. The infiltrative type produces a characteristic ulcer of irregular outline, which bleeds very readily on removal of its crust; it has a hard-rolled, elevated, nodular border and a markedly indurated base. Metastases occur through the regional lymph nodes. (After Prinz and Greenbaum.)

Illustrations: Prinz and Greenbaum, Figures 262 to 266; Thoma, Figures 382 to 387, also 390 to 404; Fordyce, Figures 73, 75, 77, 78.

Carcinomata of the tongue. Cancer may be located almost anywhere upon the tongue; although it most frequently occurs on the tip, the dorsum or the borders. The most important predisposing causes are trauma, leukoplakia and syphilis. About eighty-five per cent of cancers of the tongue are in men. Traumatic papillomata, fissures, ulcers and leukoplakia must be constantly watched as precancerous lesions. Injuries to the tongue, especially the biting of the tongue repeatedly in a particular place, due to some abnormal relation of the teeth, should be prevented by whatever procedure may be necessary. In the beginning of malignancy of a papilloma, there is a marked induration, while malignant degeneration develops at the border of the ulcer, at which point the induration is most marked. Cancer of the tongue may begin as a nodule which ulcerates, or as a disk-like, well defined, red, papillomatous or vegetating superficial lesion, with an eroded surface and with borders which lack the induration normally found in the ulcerated cancerous nodule. In the later stage, the borders are markedly indurated, the floor of the ulcer is also indurated and bleeds easily. (After Prinz and Greenbaum.)

Illustration: Prinz and Greenbaum, Figure 267; Thoma, Figures 388, 389; Hayes, Figures 237, 238; Fordyce, Figures 69, 71, 72, 74.

CONNECTIVE TISSUE TUMORS.

LIPOMATA. These are fatty tumors, more or less circumscribed, which present a soft, dough-like, inelastic sensation on palpation. They may occur almost anywhere beneath the skin or mucous membrane. Lipomata of the tongue, single or multiple, are occasionally observed.

Illustration: Thoma, Figure 241.

CHONDROMATA. These are hard, nodular, fibro-cartilaginous growths, most frequently seen in young persons and especially in structures containing bone. They have very rarely been observed in the salivary glands, and even more rarely in the tongue in connection with the median septum.

MYXOMATA. Tumors of this type are composed of mucoid or jelly-like tissue, which appear grayish or reddish-white on the surface. True myxomata are exceedingly rare, but myxomatous degenerations of connective tissue tumors is rather frequently observed.

Illustration: Thoma, Figure 305.

OSTEOMATA. Tumor-like masses composed of bone are most frequently seen in connection with bone, cartilage, periosteum or medulla. They are slow growing, firm, painless, circumscribed enlargements, which may be diagnosed with the radiograph.

Illustration. Thoma, Figures 301, 302.

MYOMATA. These are rare tumors composed of a muscle tissue.

EPULIS GRANULOMATOSA. These consist of small growths which spring from the peridental structures and are attached about the margins of the gingivae. They have been classified as "fibrous epulis," "granulation tumors," etc. They vary in size from a pea to a hazel nut or even larger, are composed of connective tissue with an epithelial covering. Occasionally tumors of this type force the teeth apart. They may have a broad base, but are often attached by a pedicle. "When they are newly formed they are generally very vascular and red and bleed easily when injured. When of long standing they become more solid and firm, often losing their inflammatory character and changing to fibroma." (After Thoma.)

Illustrations: Thoma, Figures 221 to 225.

FIBROMATA. This tumor is usually a peripheral type and may occur in any position in the mouth, or "it may arise from the periosteum of the jaws and form a tumor under the skin on the neck." This latter type may become very large. Fibromata most frequently spring from the alveolar process and may be sessile or pedunculated. Fibromata of the tongue may appear as firm, intra-muscular growths, or as pinkish pedunculated, soft masses. Very rarely a fibroma occurs intra-osseously. In the mouth, the growth may vary from the size of a pea to that of a hen's egg. It is covered with epithelium, usually of normal type, and causes no pain or discomfort, except as it may interfere with speech or mastication. (After Thoma, and Prinz and Greenbaum.)

Illustrations: Thoma, Figures 226 to 230; Blair and Ivy, Figures 145 to 148; Prinz and Greenbaum, Figure 272.

BENIGN GIANT CELL EPULIS. There are two types of benign giant cell tumors, one of which appears as a growth on the gum, springing from the peridental membrane or periosteum of the alveolar process; the other involves the bone itself and destroys the central portion. The latter is called a central benign giant cell

tumor. It is presumed that both types are caused by some form of irritation.

The growth on the gum — the epulis type — may be sessile or pedunculated. They are bluish in color, very soft and vascular, may appear as single or multiple lobes, and are composed of fibrous fibro-vascular and cellular tissue, including giant cells. These tumors, containing giant cells, are more likely to be pigmented than other epuli. They grow slowly at first and may become of firm structure, covered with mucous membrane of normal color. The size varies from that of a pea to a mass the size of a walnut. They are painless.

Illustrations: Hayes, Figures 209, 210; Thoma, Figures 206 to 211.

CENTRAL BENIGN GIANT CELL TUMOR. The tumor involving the bone may not only destroy the central portion, but will often cause a bulging of the external plates. The extent of the growth may be determined with the radiograph. The diagnosis of this type from a malignant growth is revealed by microscopic examination. It will show many multinucleated giant cells.

Illustrations: Hayes, Figures 211, 212, 213; Thoma, Figures 203, 204, 205.

KELOIDS. These are fibro-cellular or connective-tissue new growths of the derma, which occur occasionally upon the lip. They begin as small, sharply defined, hard, pinkish or whitish nodules, which slowly increase in size and assume a rounded or globular form. They may become one-half inch or more in diameter, but they never ulcerate. The surface may be traversed by dilated capillaries.

Illustration: Fordyce, Figure 51.

SARCOMATA. These are the most malignant of all tumors. They occur most frequently in early life. The cellular elements predominate and the growth depends upon the size and shape of the cells — round, spindle, giant and mixed. Sarcoma of the mouth is very rare; a few cases involving the lip have been reported. It begins as a small, red, firm, circumscribed, painless nodule, the surface of which ulcerates in a variable length of time. Metastases occur through the blood stream. A microscopic examination of the tissue is usually necessary for definite diagnosis.

Illustrations: Thoma, fibrosarcoma of mandible, Figure 233; lymphosarcoma of jaws, Figures 253, 254.

Sarcoma of the gums. This type of tumor may develop at any age, in the form of an epulis, springing from the gum, the periosteum or alveolar bone. It may be either firm or soft and is generally painless in the early stages. Its growth is rapid as compared with the benign epulis. (After Prinz and Greenbaum.)

Illustrations: Hayes, Figures 239, 240, 245, also Plate No. 11, colored.

ENDOTHELIOMATA.

These constitute a group of tumors, which are highly vascular, formed by proliferation of endothelial cells.

ANGIOMATA. These are congenital tumors, formed of hyperplastic and newly formed blood vessels (hemangiomata) or lymph vessels (lymphangiomata). A hemangioma may be a simple, flat or capillary angioma of strawberry or port wine color; or a tuberous or cavernous type, in which the lesion may be irregular or smooth, elevated and composed of a series of cavities. It may occur in any vascular structure, as the tongue, cheeks or lips. Lymphangiomata are rare. They appear early in life and occur most frequently in the tongue and lips. See macroglossia and macrocheilia, respectively. (After Prinz and Greenbaum.)

Illustrations: Thoma, hemangioma of tongue, Figure 243; of gum, Figures 245-247; Fordyce, Figures 11, 12, 13, 14.

LYMPHO-SARCOMATA. These are painless tumors of the lymph nodes, which enlarge slowly. The symptoms vary according to the location, rapidity of growth and manner of spreading. Multiple tumors form and generally grow very rapidly and infiltrate the surrounding tissue; metastasis occurs through the lymph vessels.

Illustration: Thoma, Figures 252, 253.

HODGKIN'S DISEASE. A painless swelling of the lymphatic glands of the neck, axilla and elsewhere, associated with anemia, which terminates fatally. This condition is definitely indicated by the presence of large cells with peculiar, large, pale vesicular nuclei.

Illustrations: Hayes, Figures 151, 152.

TUMORS OF THE SALIVARY GLANDS.

Tumors of the salivary glands consist of adenoma, fibroma, angioma, lipoma, sarcoma, carcinoma and mixed tumors. The latter represent the largest group. They are composed of epithelial and connective tissue elements and often contain cartilage, mucous and fibrous tissue. They occur most frequently in the parotid gland in early adult life, and are generally benign, firm and painless. However they often become cystic. They appear below the lobule of the ear, or at the angle of the jaw, as sharply defined nodules with a smooth and, at a later period, an irregular surface. The growth is very slow and may remain stationery for years. Malignancy may occur after many years, in which case the surrounding tissues may be rapidly infiltrated, and may ulcerate through the skin. (After Prinz and Greenbaum.)

CYSTS OF THE ORAL CAVITY.

Cysts are tumors containing fluid; the supporting wall is of connective tissue, lined with epithelium or endothelium. Retention cysts result from an obstruction of the passage or duct through which the secretion is normally discharged. Dermoid cysts are congenital in origin, and are the result of the inclusion of ectodermal cells in an epithelium lined connective tissue wall. These cysts contain epithelium, hair, teeth and other appendages of the skin.

MUCOUS CYSTS may occur anywhere on the mucous membrane of the mouth. They appear as small, globular, usually freely movable, faintly bluish elevations, and are filled with a thick fluid, which will be discharged if the thin outer wall is punctured. They are formed by the blocking of the openings of the mucous glands and are usually due to trauma. They occasionally reach considerable size, possibly as large as an English walnut. They are painless, except where irritated by trauma or infection.

Illustrations: Hayes, 36, 37, 38; Fordyce, Figure 8.

RANULA. This is a peculiar type of cyst. It is a soft, painless swelling beneath the mucous membrane of the floor of the mouth, containing a clear ropy fluid. These cysts are due to an involvement of the incisive glands, but most commonly to the obstruction of the excretory ducts of the sublingual gland. Retention cysts of the Blandin-Nuhn glands are also classified as ranulas. (After Prinz and Greenbaum.)

Illustrations: Blair and Ivy, Figure 295; Prinz and Greenbaum, Figure 286.

RADICULAR CYST. ROOT CYST. See Diseases of Dental Pulp and Periapical Tissues.

FOLLICULAR CYSTS. Cysts of this type are derived from the epithelium of the enamel organ of the developing tooth. They are therefore formed about the time that the corresponding teeth would normally be formed, but may not be found until later, as there may be no symptoms. They are frequently discovered in a routine radiographic examination. Thoma classifies these as: (1) simple cysts, without tooth formation; (2) dentigerous, containing a single tooth; (3) containing an odontome; (4) containing multiple tooth particles. The cause of these cysts is not clear; they represent an abnormal development within the tooth follicle.

Illustrations: Thoma, Figures 163-181; Hayes, Figures 295 to 312.

ODONTOMATA. An odontome is a tumor composed of tooth tissue in varying proportions and different degrees of development. Odontomes are usually painless; the contour of the jaw may be

abnormal, due to the size of the tumor, and they are generally diagnosed with the radiograph. The point of a sharp, steel probe, brought into contact with the mass, will detect the presence of areas of enamel or cementum, as being of greater density than bone. If the tumor is composed of a single tooth tissue, it may be so named; when all of the tooth tissues are present, it is called a composite odontome. Odontomes are generally found in the posterior portion of the jaws, upper or lower. They rest in the jaw, surrounded by a wall—presumably the persistent follicular wall of the growth. Dr. Thomas L. Gilmer reported the removal of an unusually large odontome, which measured 34 mm. x 18 mm. x 19 mm.

Illustrations: Thos. L. Gilmer, *Oral Surgery*, 1907, Figures 71 to 78; Thoma, Figures 285 to 292; Hayes, Figures 271, 272, 273.

ADAMANTINOMATA. MULTILOCULAR CYSTS. These are benign tumors of the jaws, which arise from the dental epithelium. They occur more frequently in the mandible than the maxillae. The patient will have noticed a painless swelling, usually in the molar region, which has been increasing in size very slowly. The alveolar border of the bone is enlarged; the outer plate being more affected than the inner. There is a peculiar elastic sensation on pressure. These growths begin as solid structures and later become cystic. They contain numerous cavities filled with a viscous brownish fluid. These cavities vary much in size and may coalesce. The diagnosis may usually be completed with the radiograph. (After Blair and Ivy.)

Illustrations: Blair and Ivy, Figures 159, 160.

Diseases of the Soft Tissues of the Mouth

There are a number of diseases which involve only the soft tissues of the mouth, others that occur locally in the mouth and elsewhere. The tumors which involve the mouth tissues have been presented under surgical disorders of the mouth. The dentist should carry the responsibility for the treatment of all diseases of the mouth; he should treat them himself or should be able to make a sufficient diagnosis to guide him in referring cases to specialists in other fields.

ACUTE ULCEROUS GINGIVITIS. VINCENT'S STOMATITIS. Trench Mouth. This is an infectious disease usually beginning at the crest of the gingivae about a few teeth, and may extend to involve the gingivae about many or all of the teeth, and occasionally the mucous membrane lining the lips and cheeks and the floor of the mouth. The gum about a partially erupted tooth is also a frequent source of the initial infection. It is a peculiarity of this disease that it frequently attacks the gingivae in a new location about the time when a previous lesion shows signs of improvement. It is characterized by a greyish-white membrane covering a necrotic lesion which is painful and tender and bleeds easily. In the more severe cases it is accompanied by severe salivation, a distinctively fetid breath, extreme tenderness which interferes with mastication and a typical mental depression. The membrane may easily be wiped off, leaving a raw, tender, bleeding surface, which is rapidly covered with a new membrane, and the margins of the interproximal gingivae early assume an eroded appearance, due to the necrosis of the tissue.

The local cause is infection with the *B. fusiformis* and Vincent's spirochete, and these organisms are usually found in large numbers in the lesions. The clinical symptoms of the disease are more significant than the bacteriological findings, because these organisms are frequently present in normal mouths, and may be found in pyorrhea pockets and in non specific gingivitis. Smears from the surface of an active lesion may be negative, especially if local therapeutic treatment has been given. The organisms can always be discovered by a careful search, yet too much reliance should not be placed on the microscopic study.

The disease is transmitted by direct contact and by towels, dishes, glasses, etc., and special precautions should be taken to prevent transmission. It frequently becomes epidemic in a region, especially in schools, camps, and boarding houses, and appears to vary in virulence from year to year and between different regions. It does not occur in edentulous mouths, and young adults and children appear more susceptible. It may occur in clean mouths, but unkempt mouths offer more favorable conditions for its rapid

development. It is generally believed that certain predisposing causes such as diet deficiency, metabolic disturbances and possibly smoking, are important factors in susceptibility, although the infection may occur in any mouth, especially in virulent epidemics.

Differential diagnosis is particularly important since these organisms are frequently found secondary to certain mouth diseases, and their presence clouds the diagnostic signs. This is especially true in diseases where the early symptoms resemble those of acute ulcerous gingivitis, such as diseases of the blood forming organs, and in certain dermatological diseases. In these diseases the mouth becomes more susceptible to infections, and the bacteriological report may lead to an erroneous diagnosis. Differential blood count should be made in all cases of doubt, especially if local therapeutic treatment does not bring a prompt recovery.

Only rarely may the secondary lesions of syphilis, and stomatitis due to mineral poisons, be confused with acute ulcerous gingivitis.

Illustrations: See Volume IV, also Hayes, Figures 4, 5, 6; Prinz and Greenbaum, Figures 66, 67.

VINCENT'S ANGINA. ULCERO-MEMBRANOUS ANGINA. The name Vincent's angina should be limited to the tonsil and throat form of the fuso-spirillar infection. The lesion is similar to that found in the gingival region except that the ulceration is apt to be deeper and direct diagnosis is the same. This form of Vincent's disease is frequently confused with primary and secondary syphilis and diphtheria and laboratory examinations are usually required to make the differentiation in doubtful cases. If diphtheria bacilli are recovered on blood serum cultures either diphtheria or a mixed infection is indicated. Due to the resemblance between the germs of syphilis and Vincent's disease, it may be necessary to resort to regional lymph node puncture to aspirate fluid for dark field examination. The absence of spirochetes in this aspirated fluid favors the diagnosis of Vincent's angina. The Wassermann test is not reliable at this stage of syphilis.

Illustrations Berberich, p. 135, Figure 1, also Zinsser-Stein, Figure 41, colored.

AGRANULOCYTIC ANGINA. See Systemic Diseases Manifested in the Mouth.

THRUSH is caused by a yeast-like fungus in the mouths of children and decrepit adults; it is more common in the warm months of the year. It may be first observed on the dorsum of the tongue. It may then involve the palate, the cheeks and the lips. There may be a sweetish, acid odor. The lesions are small, sometimes confluent and are covered by a flat, round white coating which can be readily removed in the early stages and the smears made from it show characteristic spores and numerous interlacing

threads, thereby supplying dependable information for diagnostic purposes.

Illustration: Moral and Frieboes, Figure 141. Colored.

APHTHOUS STOMATITIS. CANKER SORE. This lesion is characterized by the sudden appearance of single or several shallow erosions or ulcers of pin-head size or larger, very sensitive to irritation, sharply outlined with an intense red margin, while the floor of the ulcer is at times covered with a yellow or gray layer. They are most frequently located in the muco-buccal fold, but may occur almost anywhere on the mucous membrane of the mouth. They are apt to be recurrent. The solitary form is more common in adults, and the multiple variety in children, especially during the eruption period of teeth. Diagnosis is not difficult.

A rare form of this condition, known as Mikulicz aphthae, is recurrent over a few weeks and persists for possibly ten years or more in the mouths of adults, particularly women.

Illustration: Hayes, Figure 30; Fordyce; Figures 36, 37.

DECUBITAL OR PRESSURE ULCERS AND NEUROTROPHIC ULCERS. Two other forms of ulcer, which occasionally occur in the mouth, may be confused with the above; decubital or pressure ulcers and neurotrophic ulcers. The former are always associated with pressure of a tooth or denture and are essentially like the so-called bed sores; neurotrophic ulcers are likely to be related to loss of nerve function, as in palsy or paralysis.

Illustrations: Moral and Frieboes, Figure 292, colored; Berberick, p. 424, Figure 2.

HERPES SIMPLEX. HERPES LABIALIS. COLD SORE. FEVER SORE. This lesion pursues a characteristic course, beginning as numerous small vesicles more or less confluent, self-limited, and rapidly passing through this stage to form slight crusts, with some times an intermediate shallow ulcer stage. The lesions may become secondarily infected and resemble impetigo, or a primary or secondary syphilis. Impetigo is associated with characteristic lesions elsewhere on the face, while syphilis begins as a papule rather than a vesicle, lasts longer and may be differentiated by the various methods of demonstrating the *Treponema pallida*. In the mouths of adults, herpes may be confused with aphthae. However, the aphthae are usually solitary and the herpes ulcers multiple. The differentiation between herpes zoster, which is confined to one side of the mouth and herpes simplex may be difficult. The diagnostic signs of herpes zoster are given in the section on mouth manifestations of skin diseases.

Illustration: Fordyce, Figure 3.

LEUKOPLAKIA. SMOKERS' PATCHES. This is a very common painless lesion occurring in the mouths of persons past mid-life, which may involve, in order of frequency, the tongue, the

lining of the cheeks just back of the angles of the mouth, the lips, the palate and the gums. The large majority of cases occur in the mouths of men who use tobacco excessively in any form. In pipe smokers the palate is frequently involved with a keratotic white area. Leukoplakia seldom occurs in the mouths of negroes. Long continued or frequently repeated irritation of the mucous membrane, such as the biting of the cheeks, the contact of a sharp edge of a tooth with the cheek, or the pressure of any form of artificial appliance may cause this condition. The surface (of the mucous membrane) becomes pale, cloudy and slightly elevated. By degrees the tissue becomes thicker, more compact, the surface smoother and changes to gray, then becomes white and finally to the appearance of mother of pearl. There may be raised areas of thicker, keratinized epithelium. The forms are greatly varied but as a rule are sharply outlined; the lesions may be solitary, multiple or confluent and vary greatly in size and extent. There may be a feeling of stiffness of the areas, with a sense of dryness and pain on contact with hot or spicy foods.

The differential diagnosis of leukoplakia and lichen ruber planus may be difficult. The color of the two lesions may be the same. Lichen planus occurs more frequently in women, there are usually a number of small lesions arranged in a net-like pattern, and corresponding lesions are usually found upon the skin. Leukoplakia occurs infrequently in the mouths of women, the outline of the lesion is irregular and the patches are likely to be larger and solidly white, or composed of white areas of varying size.

A precancerous change is indicated when a surface which has long been smooth becomes thicker and there occurs papillary growths, accompanied by induration. Secondary and tertiary lesions of syphilis may also be confused with leukoplakia.

Illustrations: Fordyce, Figure 63; Prinz and Greenbaum, Figures 90, 91, also Plate V, colored; Hayes, Figures 49, 59, 90, 91, 92, 99, 100, 115, also Plate No. 6, colored; and case of leukoplakia followed by cancer, Figures 263, 264, 265; Thoma, microscopic section, Figure 367, tongue, Figure 372; Fordyce, Figures 62 to 71.

MERCURIAL STOMATITIS. The ingestion of mercury by persons exposed to it in their occupations, or who are receiving mercurial injections, may lead to a severe attack of ulcerative stomatitis, particularly in mouths that are not kept clean, or in which inflammations previously existed. The stomatitis may be preceded by a dark coloration due to the formation of sulfids in the mucous membrane. There may be a peculiar metallic taste; the tissues become swollen and congested; they are tender to the touch and bleed readily. This condition is likely to involve the gums most prominently. The initial inflammation may be in any position of irritation, such as the inner surface of the cheeks along the line of occlusion of the teeth. It may spread to include the tongue and

palate. The tongue may become swollen and ulcerated, particularly along the edges which are in contact with the teeth. There may be profuse salivation and foul odor. As the peridental tissues become more and more involved, the teeth become loose and their movements cause pain. The cervical glands may be enlarged. These symptoms, with the history of the absorption or ingestion of mercury, suffice to make a diagnosis.

Illustrations: Moral and Frieboes, Figure 14, colored; Hayes, Figures 2, 3; Prinz and Greenbaum, Figure 81.

BISMUTH STOMATITIS. The lesions caused by bismuth are similar to those resulting from mercurial poisoning, but are much less severe. There may be a bluish or purple line along the margins of the gingivae and in positions in which the gums may have been previously inflamed.

Illustration: Prinz and Greenbaum, Plate III, colored.

LEAD STOMATITIS. This is an occupational disease among persons who handle lead. It may also occur in persons who use cosmetics containing lead. As with mercury and bismuth, existing inflammation, due to lack of cleanliness or irritation, seems to be a necessary forerunner to this form of stomatitis. The gum margins, especially the papillae between the teeth, are most frequently involved. The inflammatory changes are slight, but the pigmentation is darker — producing a bluish black gum margin, the so-called “lead line.” The patient may notice a sweetish taste in the mouth, and may have attacks of colic, also wrist drop and foot drop. The history of the patient’s occupation, or the use of materials containing lead should support the clinical diagnosis.

Illustrations: Moral and Frieboes, Figure 18; Prinz and Greenbaum, Plate IV, colored.

X-RAY BURNS. The radiations from the x-ray tube, radium and similar radio active substances may cause lesions of the gums and mouth mucous membranes that are not at all characteristic, resembling in some cases a low grade stomatitis or burn and in others an ulcerative condition comparable to acute ulcerous gingivitis. A correct diagnosis can be reached only by a process of elimination, plus a history of exposure to such agents. With respect to effects of the radio active substances there are often changes in the bone marrow and the bone forming organs for which search should be made.

Illustration: Moral and Frieboes, Figure 10, colored.

ANGIOMA. These consist of painless, bluish red, soft elevations, occurring upon the lips, cheeks and tongue, which disappear on compression and reappear when released. They may be caused by trauma. For a more complete description of hemangiomata and lymphangiomata, see Angiomata, under Tumors of the Mouth.

Illustrations: Prinz and Greenbaum, lip, Figure 277, tongue, Figure 281; Hayes, Figures 78, 79, 80.

FORDYCE'S DISEASE of mucous membrane of lips and cheeks. See Adenomata, under Tumors of the Mouth.

GEOGRAPHIC TONGUE. Wandering rash. An intensely chronic, circumscribed, more or less circinate desquamation of the surface of the tongue. It is an infrequent disease of early infancy and childhood, but may persist for many years. The surface of the tongue is marked by clearly defined, shiny and smooth, irregular spots or circles which are slightly depressed below the level of the usually fur-covered surrounding structure. The size of the spot varies greatly; in the beginning a spot may be as large as a pea, but gradually widens into a circle and often meets another circle, with which it may intersect. These irregular map-like spots develop, giving rise to the name geographic tongue. The rash may disappear rather suddenly and, after protracted intervals, reappear as suddenly as it disappeared. Its reappearance may be due to nervousness or excitement. (After Prinz and Greenbaum.)

Illustrations: Moral and Frieboes, Figure 300, colored; Hayes, Plate No. 5, colored, also Figures 81 to 86; Prinz and Greenbaum, Figures 210, 211, 212; Fordyce, Figure 17.

BLACK TONGUE. The presence of a blackish-brown to yellow-brown, thick, soft, fur-like patch or patches on the dorsum of the tongue, often modified by a slightly bluish or more often a greenish tint, which are composed of the elongated, densely matted hair-like filaments of the hypertrophied filiform papillae, constitutes the clinical picture of black tongue. (After Prinz and Greenbaum.)

Illustrations: Fordyce, Figure 18; Prinz and Greenbaum, Figure 201; Hayes, Figure 70.

BURNING TONGUE. This is a rather rare condition in which the patient will complain of an annoying burning sensation in some particular region of the tongue. Some patients state that the burning sensation is severe, yet careful examination reveals no abnormal condition of the tongue to account for the disorder. There may be great mental distress owing to the fear of impending malignancy. The complaint may be of constant pain or of occasional recurrences. It occurs more frequently in women, who may be anemic or neurotic.

MACROGLOSSIA. LYMPHANGIOMA. This is a condition in which the tongue is enlarged as a result of the dilatation of the lymph spaces beneath the epithelium. They may contain clear fluid, or it may be hemorrhagic, due to the rupture of capillaries, and may be scattered or in patches, which cover a considerable area. These spaces may gradually enlarge and cause the surface epithelium to be thinned. Large cysts may be formed by the fusion of spaces. Occasionally the entire tongue is involved. The condition may be

congenital or may result from injury. The enlargement of the tongue, which is very slow, gradually impairs speech and causes difficulty in eating. When the tongue becomes excessively large, especially during the period of bone growth, the teeth become displaced and the palate and jaws are deformed. (After Blair and Ivy.)

PERLÈCHE CHEILITIS. A regional streptococcus infection, primarily affecting the labial commissures, usually bilateral, in older children of unclean habits and, at times, in adults. Clinically the simultaneous appearance of the disease in several members of the same family testifies to its infectious nature. A mild epidemic has been observed in schools. It is usually limited to the angles of the mouth, although it may extend far toward the center of the lips. The epithelium assumes a white, mother of pearl tinge; later it becomes macerated, thickened and changes from its white color to a brownish hue. There is a sensation of dryness, which results in the almost constant licking of the lips. The name is derived from *pour lecher*—to lick all around. (After Prinz and Greenbaum.)

Illustrations: Hayes, Figure 34, Prinz and Greenbaum, 224; Fordyce, Figure 38.

CHEILITIS EXFOLIATEVA. A persistent exfoliation of the vermillion border of the lips; a rare disease primarily observed during the period of adolescence. It usually begins with a slight redness and scaling of the lower lip, which gradually extends and involves both lips. In well marked cases the lips are covered by adherent, scaly crusts. The removal of the crusts exposes a sensitive reddened derma, which bleeds readily and is often fissured. There may be slight swelling. The symptoms are those of tension, discomfort, sometimes burning or itching and actual pain. (After Prinz and Greenbaum.)

Illustrations: Prinz and Greenbaum, Figures 227, 228; Hayes, Figure 32.

Skin Diseases; Mouth Manifestations

Diagnosis of the oral mucous membrane affections of the dermatological type is unusually difficult, as the mouth lesions in general are quite atypical, probably due to the constant presence of moisture. The correct decision is frequently determined solely by the presence of characteristic lesions on the skin. In some instances, the mouth lesions precede those on the skin or are discovered in advance, and the diagnosis can rarely be made with certainty in such cases. For this reason, the diagnostic signs of only a limited number of mouth manifestations of skin diseases will be presented. Whenever the dentist discovers a lesion of the mucous membrane of the mouth with which he is not familiar and there are what appear to be corresponding skin lesions, the patient should be referred to a dermatologist.

ALLERGY. Increasing attention is being paid to allergic reactions in all parts of the body, in addition to the characteristic manifestations of such typically allergic diseases as hay fever. The mouth mucosa is frequently involved in both types and the usual form is similar to the changes in urticaria. Puzzling varieties of gingivitis and stomatitis, especially in those persons known to be susceptible or sensitized to allergic insult, should be studied from the diagnostic point of view with such possibilities in mind.

URTICARIA, HIVES, NETTEL RASH. This condition is characterized by lesions of the dermatological type, yet it seems probable that it is usually an allergic manifestation. The lesions are typically of an edematous character. The swellings vary in size, they may be pale, almost transparent or dark red. Subjectively, burning and itching sensations may be prominent, or the swellings may be noticed in the mouth or on the skin. The swellings appear more or less suddenly, sometimes in a few minutes, and do not persist long in the mouth, as the chronic form is very rare in this region. An allergic gingivitis, not a true urticaria, may occur in women during menstruation and pregnancy and in males at puberty. Urticaria is as a rule associated with hypersensitiveness to some specific protein substance, often a component of some particular food as shellfish, strawberries, etc. Inquiry should be made as to the diet with particular reference to foods which are known to cause this condition.

LICHEN RUBER PLANUS is a relatively common, painless condition in the mouth. Whereas the lesions on the skin are very small, sharply bounded, polygonal in shape and bluish red, those in the

mouth are usually white or a bluish white and tend to the formation of peculiar net like patterns in their arrangement. They are located most often in the cheek along the elevations just between the two rows of teeth, next in order on the tongue, the lips, the palate and gingivae. On the tongue, the lesions may be in the form of solitary papules or streaks. A characteristic lesion on the skin will help in deciding on the character of the mouth lesion. The mouth lesions may persist long after the skin lesions have disappeared.

The differential diagnosis from leukoplakia is given in connection with the discussion of that condition. The mucous patch of syphilis is generally accompanied by erosion and is surrounded by a reddened area.

Illustrations: Fordyce, Figures 19, 20, 21, 22; Hayes, Figures 63 to 66, 76, 77; Prinz and Greenbaum, cheek, Figure 175, tongue, Figure 176; tissue section, Figure 177.

PEMPHIGUS VULGARIS. More than one half of the cases of pemphigus have lesions in the mouth and in many of these the mouth is involved first. Vesicles and blister-like swellings of varying size, sometimes preceded by an initial localized erythema, arise suddenly. Such phenomena may be repeated time after time over a period of years. They may involve any part of the mouth, but are especially prone to involve the cheeks and gums. Some cases begin with the simultaneous appearance of lesions in the mouth, about the navel and on the genitalia. There is a tendency to hemorrhage into the vesicles, to replacement of the vesicle by an ulcer like erosion of the mucous membrane and to secondary infection. In such cases healing is delayed and characterized by the formation of scar tissue. On the other hand simple types resolve rapidly and epithelial replacement is complete. In severe cases, nutrition is disturbed, toxins are absorbed and there is danger of aspiration of the products of tissue destruction. There is pain in speaking and eating and in later stages an unpleasant mouselike odor.

Diagnosis of the disease in the early stage, especially if limited to the mouth, is often difficult and all other diseases characterized by the formation of vesicles must be excluded, especially dermatitis herpetiformis, and conditions due to the effect of chemical and physical agents. Dermatitis herpetiformis tends to run through a definite sequence of vesicles, blisters, pustules and papules, lasting six to eight weeks with recurrences of this cycle over a long period of time up to about ten years, and beginning characteristically in the second decade of life. Subjective symptoms are itching and a burning sensation in the early stages, in many cases there is hypersensitiveness to potassium iodide, and there is no interference with nutrition or the patient's health in general, as in pemphigus.

Illustrations: Moral and Frieboes, Figure 296, colored; Prinz and Greenbaum, Figures 178, 179, also Plate X, colored.

In the severe forms of pemphigus, foliaceous and vegetans, the lesions are larger and more numerous. The vegetans is a proliferative type. The systemic manifestations are so severe as to warrant an unfavorable prognosis.

HERPES ZOSTER. SHINGLES. This is another disease, the onset of which is characterized by the formation of blisters. The lesions are located along the path of distribution of the second or third branches of the fifth nerve and confined to one side of the face and gums. The subjective symptoms are headache, limited to the same side, neuralgic-like pain, earache and even disturbances of speech or hearing and some parathesia. The vesicles may be succeeded by erosions coated with a fibrinous exudate which may be stained with blood. Enlargement of the regional lymph nodes is common and one sided angina-like lesions may be found in the tonsils. The simultaneous development of a herpes zoster along the track of a spinal nerve may assist in the identification of the affection in the mouth. The differential diagnosis from herpes simplex is presented in connection with the discussion of that condition.

Illustration: Fordyce, Figure 4.

ERYTHEMA MULTIFORME. This is another disease in which blisters may appear. Mouth manifestations frequently are an accompaniment of skin lesions, although the condition may be limited to the mouth. The lesions may be either erythematous, papular or vesiculated in character. The papular form predominates and the lesions are blue-red in the center and bright red at the periphery, or the center may consist of a small vesicle. In the mouth, these lesions tend to resolve into erosions and later actual ulcerations, due to secondary infection. The vesicles vary in size from 1 to 10 mm. or larger, the content is transparent, they bleed easily, tend to confluence and may be surrounded by an hemorrhagic border. They may be covered with a grayish membrane and the appearance may simulate leukoplakia. The most common location is the lining of the cheeks, the lips and gum margins, rarely the tongue, hard and soft palate. The manifestations of this condition are generally not severe, it persists one or two weeks and is most common in autumn and spring. Diagnosis of the mouth lesions is often difficult, as they are atypical and resemble drug erythemas such as antipyrin, the mucous patches of syphilis and the early stages of pemphigus. If typical eruptions are found on other parts of the body the identification of the mouth condition requires no further consideration.

There is a severe form characterized by a septic course, fever, enlargement of the spleen, and leukopenia, which is frequently fatal.

Illustrations: Zinsser-Stein, Figure, 35, colored; Prinz and Greenbaum, Figures 182, 183; Hayes, Figures 39, 49; Fordyce, Figure 1.

ACNE VULGARIS. This is an affection of the skin which does not involve the mucous membranes, and deserves only brief mention. The lesions frequently occur upon the face. It involves chiefly the sebaceous glands and their ducts. Papules develop at the duct outlets and the sebaceous material is retained overlong. They may become secondarily infected, forming small pustules. Diagnosis is not difficult.

LUPUS ERYTHEMATOSIS. This is an inflammatory, usually chronic disease of the skin, in which the oral mucosa is involved in a small percentage of cases. The characteristic cutaneous lesion is a mild or moderately inflamed dark red or pink, more or less infiltrated macule or patch of irregular shape and size. These are superficial and covered irregularly with grayish scales, which have horny prolongations attached to their under surfaces and project into follicular pores. The dilated pores become visible when the scales are removed. The lesions have an elevated border. Several may coalesce. They are most apt to appear on the nose, cheeks and ears; also on the scalp and the dorsum of the hands. They cause little discomfort.

About ten percent of cases exhibit oral lesions, which are likely to develop as isolated symmetrical patches on the buccal mucosa, opposite the molar teeth. They may also be present on the hard palate, gums and tongue. They begin as bright or pale red, ill-defined spots, that may join to form an irregularly shaped patch, which soon presents a variable number of very superficial erosions, partly covered with adherent moist desquamations. The area may be surrounded by a zone of dilated vessels. The patch later becomes slightly depressed in the center, presenting a grayish white atrophic area, surrounded by an erythematous edge. (After Prinz and Greenbaum.)

Illustrations: Prinz and Greenbaum, Figure 188; Hayes, Figures 47, 48; Fordyce, Figures 24, 25, 26.

Systemic Diseases; Mouth Manifestations

Since most systemic diseases have mouth manifestations in some form, the selection and grouping of the diseases to be discussed from the diagnostic point of view, presents a rather difficult problem. In the first group, not because of the frequency of occurrence, but on account of the importance of their early recognition, are the diseases of the blood forming organs, including the anemias, leukemias and agranulocytotic angina. In all of these, the diagnosis is determined on the basis of examination of the blood; counts of the cells, hemoglobin determinations and the study of stained smears—differential counts.

These will be followed by a group of bone diseases which have mouth manifestations, also a group of acute infectious and contagious diseases. Separate consideration will be given to diabetes, tuberculosis and syphilis.

DISEASES OF BLOOD FORMING ORGANS.

In connection with the discussion of several diseases of the blood forming organs, a diagnostic chart of blood examinations is presented. It will be noted that the red and white cell counts, the hemoglobin per cent and color index, also the differential counts, are tabulated for the various diseases for convenient study and comparison. The normal blood picture is given in the first column.

PERNICIOUS ANEMIA. HUNTER'S GLOSSITIS. MOELLER'S GLOSSITIS. GLAZED TONGUE. Pernicious anemia is characterized by great weakness, yellowish skin, no loss of weight, achlorhydria and a typical glossitis. The glossitis may be the first manifestation of the disease, and is therefore of great diagnostic importance. It is characterized by an atrophy of the filiform papillae of the tongue, so that the tongue initially has a very smooth, glossy surface broken only by flat, small erosions which are extremely painful, and which later cause a raw and irregular appearance. In pernicious anemia, as in the other diseases of the blood forming organs, gingivitis and stomatitis are common complications. The oral mucosa, particularly that of the palate, is pale, while the tongue is bright red. One peculiar characteristic of the tongue lesions is that they are recurrent; they disappear and recur without apparent relation to other symptoms.

Illustrations: Prinz and Greenbaum, Figures 204 to 207; Hayes, Figure 107; Fordyce, Figure 16.

LEUKEMIA. The mouth lesions of the leukemias appear early in the disease and are characteristically due to perivascular infiltrates of the white cells, especially in the mucosa and submucosa.

The epithelium is lifted, undergoes necrosis and becomes secondarily infected, thereby causing ulcero-gangrenous defects of the mouth mucous membranes, which have all the characteristics of an acute ulcerous gingivitis, when limited to the gum margins. They present the same bacterial picture in stained smears made from the lesions. Patients uniformly complain of pain and discomfort in the mouth; also of the bleeding from the gums. The breath has a foul odor, the cervical lymph glands are enlarged and may be painful. Although in some cases the patient may be gravely ill, in others the true condition is not suspected for a surprisingly long time. In both instances blood examination is the key to the correct diagnosis. The early subjective symptoms are periodic pain and burning of the mucous membranes of the mouth, accompanied by a recurrent or fluctuating gingivitis, which eventually develops into the gangrenous type. The behavior of an early agranulocytotic angina in the mouth is not essentially different from that of the leukemias, and in many instances it may resemble scurvy.

Illustrations: Hayes, Figure 8, Prinz and Greenbaum, Figure 108.

AGRANULOCYTIC ANGINA. This term is applied to a group of frequently fatal disturbances accompanied by severe ulcerative or gangrenous stomatitis, high fever, profound prostration and a neutrophil leukopenia with a relative lymphocytosis. Atypical clinical forms, *i.e.*, those without oral lesions and those with a marked tendency to hemorrhage have been observed. Agranulocytosis is not a specific disease; it should be looked upon as an abnormal reaction to sepsis. Prinz and Greenbaum.

Illustrations: Prinz and Greenbaum, Figures 110A, 110B.

SCURVY. The diagnosis of scurvy can be discussed best in connection with other diseases characterized by bleeding into or from the mucous membranes, as well as with reference to the immediately preceding group associated with a gangrenous stomatitis. Those that will be mentioned are hemophilia and purpura.

Three stages of scurvy may be distinguished, the first characterized by salivation and distention of the vascular net work of the mucosa and a tendency to hemorrhage into the swollen tissues. In the second stage not only are hemorrhages apparent but the tissues are blood tinged throughout; the teeth are enclosed by the redundant gingivæ, which are involved in small and large hemorrhages and coated with blood and saliva. The third stage is that of a severe stomatitis, with the formation of ulcers and gangrene. The gums separate from the teeth and the alveolar bone becomes involved with a fetid necrosis, the formation of sequestra and exfoliation of the teeth. This gangrenous, hemorrhagic process may and does involve the tongue and cheeks, as well as other parts of the body. Diagnosis is dependent upon the clinical picture, the demonstration of a diet deficient in Vitamin C

DISEASES OF BLOOD FORMING ORGANS. DIAGNOSTIC BLOOD EXAMINATIONS

	Normal	Pernicious Anemia	Inflammatory Leukocytosis	Myeloid Leukemia	Lymphatic Leukemia	Agranulo- cytic Angina
Red Blood Cells Total count.....	5,000,000	1,250,000	4,700,000	2,800,000	2,500,000	4,000,000
Hemoglobin Per cent.....	90 to 100	40	85-90	50	35	80-90
Color Index.....	1.00	1.50	0.95	0.98	0.70
White Blood Cells Total count.....	7,000	2,500	20,000	400,000	50,000	500-1,500
Lymphocytes Per cent.....	25.	45.	10.	1.-2.	90.	60-70
Polymorpho-neuclear Neutrophiles Per cent.....	70.	50.	85.	33.	7.	5-10
Large Mono-nuclears Per cent.....	1-2	1-2	5.	15.	1.
*Eosinophiles Per cent.....	2-3	0	0	6.	2.0
Mast Cells Per cent.....	0.5.	0	0	10.

*High up to 40 per cent or more in animal parasite involvement, as trichinosis.
Wide variations in these counts should be expected, although the relative changes are characteristic.

and the elimination of other diseases of the hemorrhagic group, as purpura. Moeller-Barlow's disease is a low grade form of scurvy in children.

Illustration: Hayes, Figure 9.

PURPURA. This disease is characterized by the spontaneous occurrence of hemorrhages into the mucosa of the mouth. The hemorrhagic areas vary in size from a couple of millimeters to a centimeter or more, and involve the cheek, the lips and border of the tongue. These hemorrhages are frequently associated with joint disturbances of a rheumatic character and hemorrhages in other parts of the body. There is no stomatitis, no ulceration and no gangrene, even in the severe fulminating variety which is generally fatal. Generally speaking, diagnosis is not difficult, as conditions with which it may be confused are quite rare.

Illustration: Prinz and Greenbaum, Plate VIII, colored.

TRUE HEMOPHILIA is an hereditary diathesis characterized by uncontrollable bleeding from very insignificant defects or injuries of the blood vessels. A fatal hemorrhage may be caused by the extraction of a tooth. Only males are involved, as the defect is carried in the germ cells of the mother, and the condition is not transmitted to daughters. In suspected cases the diagnosis is based on the history of previous serious hemorrhage in the individual, or of other bleeders in the family, and the demonstration, by testing the blood, of a very great increase over the normal time required for clotting.

Increased tendency to hemorrhage — not true hemophilia, occurs as a more or less temporary condition, associated with such diseases as jaundice, anemia and leukemia.

BONE DISEASES.

RICKETS. The identification of an active case of rickets is not difficult, since this disease is generally limited to the first three years of life and is characterized by retardation of growth, enlarged bones at the calcification centers, such as the junction of bone and cartilage in the ribs, delay in ability to walk, delayed eruption of the temporary teeth, bone deformities, pain and tenderness of the muscles, and a history of a deficiency in Vitamin D. However, a clear cut decision as to the relation of certain defects found in the mouth to rickets is difficult. Enamel hypoplasia of the permanent teeth, so commonly seen in individuals who have had rickets, may not with certainty be ascribed to rickets. The hypoplasia is much more likely to result from the associated malnutrition, or inter-current infection, such as whooping cough, measles or scarlet fever, or to an associated para-thyroid deficiency and a mild tetany. The high narrow arched palate, the mal-position of the bicuspid and molar teeth of the mandible and the crowding

of the teeth are not necessarily the result of rickets. The situation in the mouth is quite different from that having to do with certain deformities of the long bones and pelvis which may be definitely connected with rickets and which persist throughout life as recognizable entities.

Illustration: Prinz and Greenbaum, Rachitic hypoplasia of the teeth, Figure 113.

OSTEITIS FIBROSA, especially the generalized variety with cyst formation is a definite condition resulting from parathyroid hyperplasia and a high blood calcium content, disclosed by chemical examination of the blood (more than 11.0 mgms. of calcium per 100 cc. of blood). The jaw defects are disclosed as a rule by the x-ray, which shows irregular regions of rarefaction in the jaw bones. These are filled with so-called brown tumors, tissue much like that of the giant cell epulis, and cyst-like spaces. There is no very great difference between the jaw lesions of the localized and general forms, except that the former is limited to the head region, especially the bones of the face.

Illustrations: Hayes, Figures 228, 229, 230.

ARTHRITIS DEFORMANS is a disease of the joints, with a marked tendency to chronicity which may involve the temporo-maxillary articulation. In addition to symptoms referable to the interference with function (pain and limitation of motion), the x-ray film will show the bone deformity in the region of the temporo-maxillary joint, as well as similar deformities in the cervical vertebrae, and elsewhere. Differential diagnosis has to do only with other forms of arthritis, especially the specific varieties, such as tuberculosis, syphilis, etc., and even the terminal stages of some of these may assume the deforming type.

ACROMEGALY is characterized by progressively exaggerated enlargement of the bones and overlying soft structures, particularly those of the face, hands and feet, due to increased functional activity of the anterior portion of the pituitary gland. The mandible, especially, may be greatly enlarged both laterally and forward, resulting in a marked deformity. The teeth become separated as the jaw enlarges. The tongue is also enlarged; in some cases out of proportion to the enlargement of the jaw; so that it interferes with speech. In rare cases, the maxillae may also be enlarged. (After Hayes).

Illustrations: Prinz and Greenbaum, patient at 18 and 31 years of age, Figure 114; Hayes, Figures 157, 158; Thoma, Figure 105.

OSTEITIS DEFORMANS. PAGET'S DISEASE. A rare chronic disease affecting the bones. It usually occurs after mid-life, and more frequently in women than men. It causes a softening and thick-

ening of the bones, so that they lose the strength necessary to support the body. Various bones may be affected, while others remain normal. There may be enlargements of the bones of the head, including the jaws and particularly the maxillae. The radiograph will show thickening of the bone in some positions, while it is apparently entirely destroyed in others. There may be osteoporosis or sclerosis, or a combination of the two.

Illustrations: Hayes, Figures 144, 145, 146, 147; Thoma, Figures 132, 137.

ACUTE INFECTIOUS AND CONTAGIOUS DISEASES.

ANTHRAX is an extremely rare, acute infection, caused by the bacillus anthracis, which may appear in the form of splenic fever, malignant edema or malignant pustule. Either of the latter may involve the face and lips. This disease is transmitted to man by infected animals and their products.

In malignant edema there is a rapid, intense swelling, followed by localized areas of gangrene. The face and neck may be greatly swollen, and death occurs from a systemic bacteriemia.

Malignant pustule, which is the most common form, generally begins at the site of infection as an itching, red spot, which rapidly develops into a pustule, with an indurated base, surrounded by vesicles. The neighboring tissues swell rapidly and the lymph nodes are involved. Fever, rapid pulse, malaise, prostration, dyspnea and delirium may rapidly follow. In favorable cases, the constitutional symptoms subside with a sloughing off of the gangrenous area.

Anthrax frequently affects the skin and underlying tissues of the face and neck, but rarely the oral mucous membranes and the tongue, where the lesions are less pronounced than on the skin. (After Prinz and Greenbaum.)

Illustration: Prinz and Greenbaum, Cheek, Figure 157; Hayes, Figure 173.

SMALL POX. VARIOLA. This is an acute infectious and contagious febrile disease, characterized by an eruption in which macular, papular, vesicular and pustular stages develop successively upon the skin. Simultaneously pin-head or larger sized, intensely red macules appear on the palate and buccal mucosa. These become indurated and slightly papular, and their surfaces become grayish-white. As this epithelium is thrown off, a circumscribed erosion remains. When the tongue is involved, it may become greatly swollen. (After Prinz and Greenbaum.)

Illustration: Moral and Frieboes, pustules on hard palate, Figure 171.

SCARLET FEVER. Scarlatina. This is an acute, extremely contagious streptococcal disease, characterized by high fever, sore

throat and a bright red, generalized, punctate, cutaneous eruption, with subsequent abundant desquamation.

The throat is sore, red, swollen, and not infrequently covered with an exudate. The tonsils, soft palate, buccal mucosa and gums may be involved. At the beginning of the attack, the tongue is heavily coated with a grayish fur, through which numerous red, enlarged papillae are scattered. In a few days the coating is desquamated, leaving a red, raw looking surface, known as the strawberry or raspberry tongue of scarlet fever. (After Prinz and Greenbaum.)

Illustrations: Moral and Frieboes, tongue Figure 161, throat Figure 162.

INFLUENZA. Oral manifestations of influenza occur in a limited number of cases. The mucous membrane is edematous, of a bluish-red color, and succulent to the touch. The lips are somewhat enlarged, with slight cracks or erosions, which bleed when stretched. The tongue is usually covered with a grayish fur. Numerous grayish or yellowish infiltrated areas appear upon the mucous membrane and especially about the tip and borders of the tongue, which ulcerate within a few days. (After Prinz and Greenbaum.)

Illustration: Prinz and Greenbaum, Figure 158.

TYPHOID FEVER. In the early stages of typhoid fever, the tongue is usually moist and coated with a grayish fur, but is bright red at its edges and on the tip. During the most serious febrile state the lips are often dry and the gums, teeth and lips are covered with heavy sordes. There may be a severe ulcerative stomatitis with shallow excoriations anywhere in the mouth.

Illustration: Moral and Frieboes, ulcers of mouth, Figure 151.

MEASLES. The only constant lesions of measles are those of the skin and mucous membranes, chiefly of the respiratory tract. An early sign of the greatest diagnostic value is the buccal eruption known as Koplik's spots, consisting of bluish white specks upon a red ground. In most cases, they appear only on the inside of the cheeks, opposite the molar teeth, although they may be present on almost any part of the buccal mucous membrane. Strong sunlight is generally necessary to see them. (After Holt and Holland.)

Illustrations: Moral and Frieboes, Figure 156; Berberich, p. 513, Figure 3; Prinz and Greenbaum, Plate I, colored.

DIPHTHERIA. An acute infection caused by the Klebs-Loeffler bacillus, characterized by the formation of a fibrinous exudate upon the mucous membranes, especially of the throat and respiratory passages, together with constitutional symptoms due to the absorption of toxins. The disease is generally limited to children.

The affection in the mouth is usually a secondary extension from the fauces to the soft palate, tongue, gums, cheeks and lips.

The lymph nodes are enlarged and painful. The breath is very offensive.

Illustrations: Zinsser-Stein, tonsils, Figure 40; Berberich p. 501, Figure 1; Prinz and Greenbaum, lip, Figure 153, gum of lower jaw, Figure 155.

EPIDEMIC PAROTITIS. MUMPS. An acute infectious disease, characterized by swelling of the parotid gland, which is painful. There is usually considerable edema of the adjacent tissues. The pain is increased by movements of the jaw, by pressure and sometimes by salivary stimulation, as by acids in the mouth. As a rule, one parotid gland will be involved a few days in advance of the other. The other salivary glands may be involved.

DIABETES.

More than seventy-five per cent of persons affected with diabetes have mouth lesions, either in the form of stomatitis, resembling acute ulcerous gingivitis, or a malignant pyorrhea. It is estimated that about two per cent of the population of the United States is diabetic. Of these persons probably more than eighty-five per cent are more than fifty years of age, and in a large number of cases the patient sees a dentist more frequently than a physician. Therefore, the dentist should refer to their physicians all patients who present mouth symptoms which suggest diabetes.

The general symptoms of diabetes are marked thirst, polyuria and loss of weight. In cases uncontrolled by diet or insulin or both, the gums are hypertrophied, red or purple, and frequently ulcerated with necrotic surfaces, while the breath has a sweetish, fruit-like odor. Stained smears from the diseased gums resemble those made from the lesions of acute ulcerous gingivitis. In patients under treatment, there is little or no recession of the gums and but little pus, although the mucous membranes are spongy and inelastic. However, the pockets are deep and there is a marked loss of alveolar bone. Extractions of teeth in this latter group will be found to have been numerous, either because of looseness of the teeth or to eliminate a suspected oral focus of infection in the treatment of conditions resembling neuritis, neuralgia or arthritis. The mouth conditions described above should suggest the possibility of diabetes and the necessity for the examination of the urine and blood, the former for the presence of sugar and the latter for the retention of sugar. In borderline cases a sugar tolerance test may be helpful. Once the attention is directed to diabetes, the diagnosis is usually easy.

TUBERCULOSIS.

Tuberculosis is rare in the oral cavity, except in cases of advanced pulmonary phthisis and usually secondary to lesions elsewhere, as in the lungs or on the face.

Lupus vulgaris, the slowly progressive tuberculosis of the skin, may be primary on the mouth mucous membrane or secondary to the lesions of the face. It usually arises simultaneously in many more or less widely separated places, as the vermilion border of the lips or the soft palate, and runs a very chronic course similar to that on the skin.

Illustration: Prinz and Greenbaum, Figure 145.

Primary tuberculosis of the mouth other than lupus is usually solitary and somewhat resembles the so-called anatomical wart in some instances and is ulcerative in character in others. The latter condition occurs in children at the time of the eruption of the permanent teeth and is located on the gingivae or in the pulp of a tooth exposed by caries. This ulcerative form usually extends to the regional lymph nodes with typical involvement of them.

Secondary tuberculosis is usually transmitted to the mouth from an open lesion in the lungs or in the generalized form through the blood stream. In the latter the lesions are usually miliary in character. The lesions are generally ulcerative, although they may be of the infiltrative type.

Diagnosis is based on the clinical appearance, especially the demonstration of miliary tubercles in the ulcer or beneath the surface of the mucous membrane, the absence of board like induration and the presence of pain, which is often severe, the characteristic enlargements of the regional lymph nodes and typical tubercles in stained sections of excised tissue or acid fast bacteria in smears made from the lesions. Diagnosis is always a difficult problem, since the lesions must be differentiated from syphilis, carcinoma, other infections and injuries.

Illustrations: Thoma, Figure 81; Fordyce, Figures 28 to 33; Hayes, tongue, Figures 93, 94, 95, 96; Prinz and Greenbaum, Figures 146, 148, also Plate IX, colored.

SYPHILIS.

The primary lesion of syphilis — the chancre — may occur anywhere on the body where conditions favor the penetration of the spirochetes beneath the body covering. From such a lesion the organisms are disseminated to all parts of the body, and within a few weeks after the beginning of the chancre, eruptions may be found on the skin and mucous membranes parallel with other changes, all of which are grouped under the name of secondary syphilis. These generally subside, with or without treatment, and there follows a latent period of variable length, often many years, after which syphilis reappears in the third or tertiary form. The manifestations of this stage are usually local, severe and destructive, in marked contrast with the mild character of those of the first two stages. From the diagnostic point of view, it is important that all three types may be and are found in the mouth, especially

the second in more than half of all cases of syphilis. Both the primary and secondary lesions of syphilis are extremely contagious, and their diagnosis is, therefore, of great importance to the dentist.

Illustration: Prinz and Greenbaum, *spirochæta pallida*, Figure 115.

Chancre. Extra-genital primary lesions constitute slightly less than ten per cent of all cases. Fournier's figures have been widely quoted. Of 1,124 cases of chancre found on other parts of the body than the genitalia, 849 were in the head region. Of the latter, 567 were located on the lips, 75 on the tongue, 69 on the tonsils, 11 on the gums, and one on the cheek lining. The lesion appears two to four weeks after exposure and is either eroded or papular in form. In either case there is more or less induration, which is peripheral or deep seated, often both, and more extensive in the papular form. On the lip the chancre is usually eroded and ulcerative in character. The base is distinctly indurated and the regional lymph nodes are usually swollen and very firm. Diagnosis of a typical chancre without secondary complications is not difficult, if the clinical course, the absence of pain, the localized induration and the characteristic involvement of the regional lymph nodes are given due consideration. Lesions on the tongue, tonsils and gums are, as a rule, not easily diagnosed. Search should be made for *typical spirochetes* in the suspected chancre, using the dark field microscope, also in fluid aspirated from the swollen lymph nodes. The Wasserman and Kahn tests of blood serum cannot be used at this stage of syphilis.

The principal secondary lesion of syphilis is the mucous patch, which occurs on the lips, particularly in the commissures, and on the dorsum of the tongue.

Secondary syphilis. Mucous patches. These appear four to six weeks after the onset of the chancre. Their form and appearance varies from that of a slightly elevated, faintly shining region, a little redder or grayer than the surrounding membrane, to a markedly hypertrophied lesion with a characteristically rough surface. All, especially the latter, may become eroded and secondarily infected with formation of superficial or deep ulcers. In the deep form there is extensive tissue destruction, and this form is most often found in such mouths as are grossly neglected. Diagnosis is based on careful observation, the effect of anti-syphilitic treatment, the demonstration of *typical spirochetes* in the lesions and on the result of either the Wasserman or Kahn blood serum tests, in the latter phases.

Tertiary syphilis. Gumma. The third stage develops rapidly, especially the gumma, and tends to spread so that a considerable amount of tissue undergoes coagulation necrosis. Secondary softening takes place rapidly after it has been initiated so that in a relatively short time, even a few days, a large defect may be formed, as in the palate with perforation into the nose and loss

of a major portion of the soft palate. However, in the tongue and lips the process is usually much more chronic and associated with less necrosis and more fibrosis, with an irregular and diffuse infiltration of the fibrous tissue. The tongue may therefore become deeply furrowed (sclerosis of the tongue) or may have regions of localized hyperplasia (*lingua lobata*). All third stages are prone to ulceration, followed by a type of healing which produces rather typical radiating scars. There is a form of leukoplakia which may result from the changes in the tissues caused by syphilis and it is significant that these and other chronic alterations are likely to serve as the foundation for the development of cancer. As previously stated local pain is not common in syphilis, and in contrast with the first two stages, the third stage lesions are only very feebly contagious. It is very difficult to demonstrate the presence of spirochetes in them. Diagnosis is quite similar to that in the second stage, and the differential diagnosis with respect to tuberculosis and cancer is of the greatest importance, especially the distinction between syphilis and carcinoma. Examination of excised tissue by a trained pathologist may be required to establish a diagnosis.

Little is definitely known about the incidence of syphilis. It has been estimated that it is as high as 25 per cent of the population in certain parts of the United States (Mississippi) and less than 5 per cent in other places (rural districts in the northern states). Large urban centers, as New York and Chicago, may have an incidence of 10 per cent.

Illustrations: Chancre: Zinsser-Stein, Figures 2, 3, both colored; Moral and Frieboes, lip, Figure 192, colored; Prinz and Greenbaum, lip, Figures 116 to 119; Hayes, Plate No. 7, colored; Fordyce, Figures 46 to 50, 52.

Mucous patches; Zinsser-Stein, Figures 9, 10, both colored; Moral and Frieboes, lip, Figure 208, colored; Prinz and Greenbaum, papules on lip and tongue, Figures 122 to 125; Fordyce, Figures 34, 35, 39 to 43.

Gumma; Zinsser-Stein, perforation of palate, congenital type with "Hutchinson teeth," Figure 29, colored; Moral and Frieboes, Figure 234, colored; Prinz and Greenbaum, perforation of palate, Figure 138; Hayes, gumma of lip, Figure 119; perforation of palate, Figures 121, 122; gumma of palate, Figures 123, 124; gumma of jaw, Figures 125, 126; osteomyelitis of jaw, 127, 128; deformities of nose, Figures 132, 133; Fordyce, Figures 54 to 58.

HEALTH, NUTRITION AND HYGIENE

ILLUSTRATION: FIGURE 7.

BY CLARA M. DAVIS, M.D.*

THE inclusion of a chapter on Health, Nutrition, and Hygiene in a work of this character is predicated on, indeed almost demanded by, the emphasis throughout these volumes on the conservation of teeth. And in considering their relation to the development and maintenance of sound teeth we shall be chiefly concerned with their operation during the earlier years of childhood. For the early maturity of the teeth limits strictly the time in which health and nutrition can function for the production of teeth that are well formed and well calcified, and it is in early childhood too that the vicissitudes of disease and malnutrition are, in general, most promptly reflected in decreased resistance of body tissues to untoward influences and especially to infection.

EFFECT OF HEALTH AND NUTRITION ON THE TEETH.

In just what manner and to what extent the teeth, as living members of a living body, are affected by its health and nutrition are questions that as yet are, for the most part, unanswered. What developmental defects are due to childhood disease, what to endocrine disturbances, what to faulty nutrition? To what extent if any is caries a nutritional disorder? And, if there be dietary factors in the etiology of caries, are they of the nature of a specific deficiency of one or more food factors; of a general insufficiency of food, i.e., undernutrition rather than malnutrition, or are they merely productive of deleterious effects in the mouth, as possibly, for example, sugars? The answers to these questions concern pediatrics almost as much as dentistry and the approaching solution of the problems involved foreshadows the time when the teeth of children may prove a valuable yardstick for the appraisal of the child's past health and nutritional history.

The relation of health, nutrition and hygiene and of the lack of these to conditions in the dental field can not be evaluated without a reasonable understanding of the effects of disease, inadequate nutrition and lack of proper hygiene on the body as a whole, particularly during the childhood period. There can be no question of the bearing which the general physical condition has upon the diseases of the teeth and their supporting structures.

EFFECT OF DISEASE DURING THE DEVELOPMENT OF THE TEETH.

There is, however, no need for the excursions into the field of the unsettled and controversial to establish the importance of health

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and nutrition in the life history of the teeth, for the characteristic hypoplasia of the teeth presents not only definite evidence of diseases or periods of malnutrition during tooth development, but also records the age of the child at the time of the illness.

AFTER ERUPTION, it is true, these hazards to gross structure no longer exist or, rather, they have been exchanged for the hazards of environment — mouth, gums and saliva — whose favorable or unfavorable conditions are largely determined by the character of the foods and bacterial flora that come in contact with them and by the state of health and nutrition of the individual. The rapidly developing multiple caries in hitherto sound teeth appearing after such diseases as typhoid fever and such nutritional drains as pregnancy and lactation bear witness to the continued susceptibility in later life to disturbances in the general health and nutrition, diet and hygiene. The characteristic changes in the teeth and their supporting tissues that accompany certain diseases are so well known and their descriptions are so readily accessible in both dental and medical literature that they need no repetition here.

Temporary teeth. It is interesting to note that gross structural defects in the temporary teeth are extremely rare. Their generally perfect form, notwithstanding serious illness or malnutrition of the mother during pregnancy, are evidence of the fact that the foetus is protected from the nutritional damage often suffered by the mother.

EFFECT OF ILLNESS AND POOR NUTRITION. It is desirable that we know, for the purposes of preventive dentistry, the effect of illness in general and of the milder grades of poor nutrition on the *quality* of body tissues as they are built, on their maintenance after maturity, and on the “vital resistance” by which they survive with a minimum of damage and a maximum of repair, the vicissitudes to which life inevitably exposes them.

Illness and/or poor diet, qualitatively or quantitatively inadequate, account for an overwhelmingly large proportion of the damage to both structure and vital resistance of tissues that children suffer in their early years.

In appraising the damage done by illness to the quality and vital resistance of developing tissues, not only must the character of the disease itself be kept in mind, but also its severity and duration, the age and nutritional status of the child at the time of its onset, and even the changes that illness entailed in the child's diet. Growth in stature and in the size of vital organs is a continuous process at an ever decreasing rate throughout childhood. Inexorably, in sickness as in health, in the undernourished as in the well nourished, both general growth and the development and calcification of the teeth go on, *but little affected in rate*, save as endocrine function may be impaired, according to the schedule laid down for

the species and *despite paucity and poorer quality of building materials*. Illness and poor nutrition, then, fail to appreciably reduce the demands of growth for food materials while seriously limiting the possibility of meeting them. The rapidity of growth in young children also precludes the laying up of such important reserves of minerals, proteins, and of some of the vitamins, as are present in the adult body and which help to tide over times of scarcity. Generally speaking, then, *the younger the child, the more serious are the effects of illness, malnutrition and poor hygiene* on the structure, quality, and vital resistance of tissues and the more quickly are the effects produced.

LOSS OF APPETITE and reduction of food intake in consequence are, of course, common to illness at all ages. Little children, however, are far more prone to vomiting and diarrhoea with any and every kind of illness than are older children and adults, so that, in the very young, losses of both ingested food and of important minerals from the body itself occur from these causes that may be serious to a degree quite out of proportion to the nature of the illness itself. Only the mildest and most transitory of illnesses in childhood fail to inflict nutritional damage on the growing organism. Moreover, many diseases inflict *direct* damage, (more often of a weakening than of a destructive nature), on both growing and mature tissues through the production of soluble toxins and alterations in the blood and other vital fluids. Naturally, diseases vary widely in their capacity for damage, whether toxic or nutritional, so that for the proper evaluation of the importance of illness in the history of an individual, some knowledge of this aspect of them is desirable for the dentist as well as for the physician.

IMMUNITY AND VITAL RESISTANCE ARE DIFFERENT AND UNRELATED.

At this point it seems worth recalling that *immunity* to specific contagious and infectious diseases and *vital resistance of tissue* are separate, distinctly different, and, so far as our present knowledge goes, unrelated phenomena. Immunity to the "taking" of infectious disease and especially to such as tend under favorable circumstances to become epidemic, is not a by-product of good health and vital resistance of tissues, but a racial, familial, or personal acquisition, either from actual experience of or repeated slight exposures to the specific disease, or the harboring of the parasites in a form too attenuated to produce the illness. Thus the miserably nourished child of the slums often has a higher immunity to diphtheria and to tuberculosis than the rosy-cheeked, well-nourished farm child. Optimistic nutritionists from time to time have, it is true, encouraged by implication, if not directly, the view that healthy children properly fortified by abundant vitamins had thereby a higher immunity to colds and other contagions, but the evidence

thus far adduced in support of the contention is, to say the least, unconvincing. On the other hand, the gradual acquiring of immunity to contagions and infections that are endemic, the virulence of such as are newly introduced into a community, and the constant modifications by way of gradual adaptive changes in hosts and parasites by which such diseases tend to become milder once they have become established in a population, are all well-attested phenomena of epidemiology. This, of course, is not to say that health and nutrition have no bearing on what happens to a child once he has acquired an infection or contagion — far from it — for a fatal result, or complications that drag out its course are often contingent upon the child's state of health and nutrition at the time of onset and thus not infrequently determine the outcome.

The fact that a child has had (if without complications) such diseases as simple colds, chickenpox, measles, mumps, or even acute primary *lobar* pneumonia — all of which are self-limited and of short duration — may be disregarded for all practical purposes, as these do not warrant the assumption of either general tissue damage or lowered vital resistance, other than the most immediate and temporary.

DISEASES OF CHILDHOOD WHICH CAUSE TOXIC AND NUTRITIONAL DAMAGE.

What then are the diseases that in early childhood leave behind them a train of toxic and nutritional damage and are therefore of importance in a child's history regardless of his appearance or weight?

DIARRHOEAL DISEASES AND SEPTIC STATES; PROLONGED LOWERING OF VITAL RESISTANCE. First in frequency during later infancy and early childhood are diarrhoeal diseases and septic states, whether primary or secondary to some milder illness. Loss of weight and strength are extreme, appetite fails almost completely, convalescence is slow and protracted, and lesions of gums, lips and tongue that make the taking of food painful are common. Their toxicity is often made apparent by the occurrence of hyperpyrexia, convulsions, or meningeal symptoms. How serious is the damage to vital resistance is shown by common hospital experience, viz., that many of these little patients recover from such diarrhoeas or septic illnesses and are discharged only to be readmitted some months or a year later ill with an apparently mild pneumonia or other infection that rapidly proves fatal.

A study made by Louis I. Dublin on Typhoid Fever and its Sequelae,¹ some twenty years ago, covered the 1936 cases occurring among the policy holders of the Metropolitan Life Insurance Company during 1911. After the elimination of 376 for uncertainties

¹Amer. Jnl. of Public Health—Jan. 15, 1915.

in diagnosis and 140 deaths, there remained 1428 survivors. These were distributed by sex, color and ten-year age periods and compared with similar groups taken from the mortality tables for the same years. From such comparisons it was found that in the series the expected death rate was 26.45 and the actual death rate 54, the conclusion being that during the first three years after typhoid fever, the mortality is twice the normal rate — tuberculosis, heart disease and pneumonia being the most frequent causes. So clean-cut a demonstration of the prolonged lowering of vital resistance that occurs after serious toxic and debilitating diseases is of the greatest significance with respect to other diseases of the same general character.

CHRONIC DISEASES OCCURRING ABOUT THE THIRD OR FOURTH YEAR, OR LATER. The same variation in capacity for general tissue damage and lowering of vital resistance is seen among the more chronic illnesses that show a predilection for children from three or four years on. Tuberculosis may cause greater damage to infected bones and joints than occurs in rheumatic and other non-tubercular chronic arthritides, yet the untoward effects of the diseases of the latter group on general nutrition, and on liver, kidneys and heart, are much more serious, and there is greater evidence of toxicity in the depression of appetite, spirits and activity.

RELATION TO DENTAL CARIES. Interestingly enough the dental status of the two groups shows parallel differences. As is shown by the dental records of the Children's Memorial Hospital in Chicago, the children suffering from tuberculosis of bone or joints, although they have no immunity to caries, differ little if at all from the general run of the dispensary and hospital clientele. On the other hand, rampant caries in teeth of both the temporary and the permanent dentition, with frequency of periapical infections is a common accompaniment of the diseases of the arthritic group. Opinions differ widely as to the etiological relation of such dental findings to these conditions. To the writer they seem to be concomitant manifestations of the diseases, rather than foci of infection that are responsible for or initiate the diseases. We may also suspect that nutritional and hygienic factors are of importance in the etiology, from the circumstances of their occurrence, viz., that cases are uncommon in private practice, that multiple cases in the same family are by no means rare, and that their frequency is greatest in families whose economic status, diet, living quarters and hygiene are poor as judged by present day standards.

TREATMENT IS MEDICAL; RESTORATION TO HEALTH IS NUTRITIONAL. Such in general are the types of diseases most important in the child's history as pointing to the probability of both toxic and nutritional damage with lowering of vital resistance of tissues

and weakened powers of repair as aftermaths. The treatment of the diseases themselves belongs to medicine; the management of their untoward results with a view to the most prompt and complete restoration to their optimal state of body fluids, cellular functions and vital resistance and protection of the child from falling an easy victim to further ills, falls largely in the field of nutrition.

NEED ABUNDANT DIET FOR LONG PERIOD. It scarcely needs saying that these children for a long period after apparent recovery stand in need of an abundant diet of the most nourishing foods, with a minimum of starches and sugars which are merely energy supplying and quick satisfiers of hunger as well. It seems equally plain that for rapid regeneration, repair and functional restitution of vital cells, the most important elements to be furnished by the diet are the high grade proteins (eggs, liver, kidneys, red meat, cheese and milk) and the minerals. For protoplasm has no other absolute essentials than proteins, minerals and water.

AFTER HEMORRHAGE AND DEHYDRATION. It will also be recalled by the reader, that after hemorrhage, and in the dehydration that so often occurs in the course of infantile diarrhoeas of infants and other serious diseases of children and adults alike, Ringer's solution is administered subcutaneously or intravenously, not only for replenishing fluids but for replacing mineral losses. And so vital are both water and minerals to the survival of protoplasm, that once these have been replaced, life can often be saved.

IRON AND CALCIUM PHOSPHATE. The practice of prescribing iron for the anemia of convalescence, rather than awaiting its slow reaccumulation from diet, is a long established and sound procedure, and after illnesses in the growing period it would seem to be equally rational in the interest of growing bones and teeth in process of calcification, to administer calcium and phosphorus with the same end in view, viz., the almost immediate provision of adequate supplies of these elements instead of waiting for their slow accumulation from diet. We hear much about calcium, relatively little about phosphorus, yet it is important to keep in mind that it is with calcium phosphate and not calcium or any other of its salts that calcification of teeth and bones is accomplished. The administration of calcium and phosphorus in the form of di-basic calcium phosphate is easy, cheap and requires little if any oversight. Veterinarians use it extensively as an adjuvant to the diet of pregnant, lactating and growing animals, and no contraindications to its use in the young have been found. It is given regularly in doses of two drams a day to almost all of the patients in the Orthopedic ward of the Children's Memorial Hospital and, with or without codliver oil, would seem to have a place in the therapy of convalescent children who have suffered nutritional damage.

LOWERED RESISTANCE MAY BE CONDUCTIVE TO OTHER DISEASES. The diseases that have just been discussed are among those whose characteristic symptoms and lesions are produced by external agents — chiefly infections — although for such of these as are not in the contagious or epidemic group to become operative, a prior lowering of vital resistance by cold, exposure, poor nutrition, unhealthful living quarters, or by frequent colds or other infections in themselves relatively harmless, or even by endocrine inadequacies, may be necessary. They present, however, none of the characteristic features of the deficiency syndromes or endocrinopathies.

NUTRITIONAL DEFICIENCY SYNDROMES.

The nutritional deficiency syndromes, whether due to lack of some particular food element in the diet or to chronic general undernutrition, are of especial interest to the pediatrician and to the dentist not only because of their effects on the building and maintenance of body tissues, and such processes as calcification, but also because no other ills to which the body may be exposed can be so simply, so surely and so completely prevented, or when they do occur can be combatted by measures so prompt, so easily applied and so truly specific.

DISEASES DUE TO LACK OF PARTICULAR VITAMINS. Of these nutritional disturbances, those best understood, as yet, are the deficiency syndromes arising from lack of a particular vitamin, viz.,

Keratomalacia, softening and ulceration of the cornea due to deficiency of fat-soluble Vitamin A (necessary for growth and the integrity of the epithelial linings of the mucus tracts and the epithelium of the skin).

Rickets, in which deficiency of fat-soluble vitamin D plays such an important part.

Beri-beri, due to deficiency of water-soluble vitamin B₁ (the anti-neuritic vitamin).

Scurvy, due to lack of water soluble vitamin C.

Sterility, due to lack of vitamin E.

And while no definite syndrome has been described for lack of vitamin G (B₂), it is generally accepted that this water soluble fraction which has been isolated from its companion B₁ and whose sources are the same, is necessary for growth, though whether its deficiency is indeed the cause of pellagra is still a matter of dispute.

We are accustomed to think of rickets as a bone disease, scurvy as a haemorrhagic disorder, beri-beri as a polyneuritis, etc., but that is hardly more correct than the classification of measles and small-pox as skin diseases which was not uncommon in text-books of 125 years ago.

RICKETS. Thus in rickets, a disease of especial interest to dentists since its effects on developing teeth are often seen, it is not the teeth and bones only that are affected but also every other system of the body: respiratory (recurring attacks of bronchitis and increased susceptibility to broncho-pneumonias), gastro-intestinal (frequent attacks of diarrhoea), nervous (convulsions), muscular (hypotonia) and, as is too little appreciated, some of these symptoms usually appear before skeletal changes are discoverable even with the X-ray. The characteristic bone changes of rickets are, of course, seen only in growing bones while the epiphyses are still ununited to the shaft and, with the rare exceptions of cases termed late rickets, in the first year. Nevertheless, striking disturbances in calcium, phosphorus and vitamin D metabolism do occur in adult life as in osteomalacia. Therapy promptly controls and repairs the damage to bones, but the ill effects of rickets on vital resistance of tissue persist long after the healing of the bone lesions, and children who have had this deficiency disease remain "delicate" for a longer or shorter number of years depending on the adequacy or inadequacy of their subsequent feeding.

SCURVY. That the *erupted* teeth may be loosened and even fall out in scurvy (vitamin C deficiency) is, of course, one of the most striking and most widely known features of the disease, but since the characteristic lesion of scurvy is haemorrhage, chiefly subperiosteal, though in severe cases sub-serous, sub-mucous, or even intra-cranial, it follows that the supporting tissues rather than the structure of the teeth themselves bear the brunt of the damage. So far as is known to the author no characteristic and permanent deformities of bones and teeth have been recognized as following uncomplicated scurvy in children. But in these, as in the other deficiency diseases, and more markedly the younger the child, among the more serious features are the loss of appetite and tendency to diarrhoea in even the mildest cases.

BERI-BERI. The degenerative changes in beri-beri (vitamin B deficiency), too, affect most of the tissues of the body and the changes in the gastro-intestinal system are even more pronounced than in the nerves, although clinically the neuritis occupies the foreground to such an extent that it has attached the appellation "anti-neuritic" to vitamin B.

NEED SIGNS OF MILD DEFICIENCIES, AND LONG CONTINUED MINOR DEFICIENCIES. These diseases — rickets, scurvy, beri-beri and keratomalacia, which is extremely rare in this country — are the results of outspoken and rather severe grades of vitamin deficiency. All are clean-cut, clinical syndromes easily recognizable and either are, or are in a fair way to become, medical rarities. Our present need, however, is more information, not only on the clinical signs and symptoms of deficiencies too mild to produce these well-known

syndromes but also on the effects of long continued minor deficiencies of these vitamins in later life. Much work has been done along these lines and there seems little reason to doubt the deleterious effects of even small deficiencies on cell health, function and vital resistance, that make the individual more vulnerable to disease in general. But, that such small deficiencies are the specific etiological factors of other diseases than rickets, beri-beri and scurvy, which like these latter can therefore be prevented or cured if need be by administration of the vitamin, has not yet been shown. The claims made, for example, in behalf of vitamin A as a preventive of colds, and for vitamin C as a preventive and arrester of caries lack much of being substantiated in a way to bring general conviction and acceptance.

OTHER IMPORTANT DIETARY DEFICIENCIES. There are, however, other dietary deficiencies as important, doubtless, as those of the vitamins, if not so dramatically in the limelight of present interest. The number of food elements thus far discovered to be necessary for sound body-building and maintenance is not less than 35, viz., 9 minerals, 18 amino-acids from which in varying combinations the different body proteins are built, 6 vitamins, and several fats and carbohydrates. Certain of these as for example, the amino-acid glycocoll, can be synthesized by the body itself, but with the exception of those that can be so synthesized (whose number and identity is not yet known), all must be supplied by the diet and the lack of *any* in the diet constitutes a true dietary deficiency. Of these, protein and mineral deficiencies and even deficiencies of fat are not only more important and common but easier to bring about under our present dietary habits than is deficiency of carbohydrate. The need for the latter is minimal, as shown by the diets of such carnivorous people as the Esquimaux, while the fact that both carbohydrates and fats are predominately energy supplying foods and as such can to a large extent replace each other in the diet, makes a deficiency of either more difficult to bring about.

GROSS DEFICIENCY OF PROTEIN gives rise to a clinical syndrome as clean-cut as that of rickets or scurvy, viz., sudden appearance of general marked edema, without heart or kidney disease. From its frequent occurrence in the Central European countries during the World War, it is often referred to as "war edema"; in infant wards in this country it is usually called "nutritional edema." And there is every probability that in addition to this disease caused by a general deficiency of protein and easily curable by the addition of common protein foods to the diet, we shall in the near future be able to identify clinically deficiencies of some, at least, of the constituent amino-acids, for the proteins we use in our diets differ widely in their relative content of the different amino-acids and it is on the basis of whether or not the various *food* proteins furnish the amino-acids in amounts most nearly corresponding to the needs

of the human body that they are rated as biologically high or low grade proteins.

PROTEINS IMPORTANT FOR CHILDREN AND FOR MINERAL METABOLISM. How important are abundance and quality of protein in the child's diet becomes apparent even on most casual consideration of the question. As H. C. Sherman says "the proper selection of protein is important in the feeding of children, who differ from most other animals in that their period of growth is so much longer than their suckling period. Even the child that is nursed for a year and attains three times his birth-weight before weaning will have much the greater part (probably five-sixths) of his growth to make on food other than milk. By the time his growth is complete he will probably have about twenty times the body weight and possibly more than twenty times the body protein with which he was born." To this might be added that his heart, his blood vessels, his brain and all his glandular organs will be made predominantly of protein. The importance of protein for mineral metabolism should also be mentioned, it being necessary for the retention and storage of minerals. It is in the muscles, glandular organs and blood that minerals are found in abundance, and in bone the protein of which provides the matrix necessary for calcification. On the other hand, storage of minerals in connection with fats is minimal.

MINERAL DEFICIENCIES, unconnected with protein deficiency, such as cretinism, simple goitre and other forms of hypo-thyroidism, due to lack of iodine, and infantile tetany due to lack of calcium are well known, while the form of collapse in stokers, packing house workers and others, familiar under the name of "heat prostration" and due to the deficiency of sodium caused by the loss of sodium chloride from excessive sweating, is almost equally so. All are promptly relieved by supplying the lacking mineral, iodine, calcium or sodium in the form of one of its common salts.

CARBOHYDRATE DEFICIENCY. As mentioned before the need for carbohydrate is minimal and amply covered in any and every American diet. Its deficiency syndrome is thus rarely, if ever, seen in the conditions of ordinary life and health, but was not infrequent in the early days of the treatment of diabetes with insulin. For its description under the title of Hyperinsulism, any modern textbook on general medicine may be consulted; proof of its nature is in the lowering of the blood sugar that is invariably present and in its successful treatment by the administration of carbohydrate.

These then are the proved and well-known deficiency syndromes. Such others as there are or may be, remain, for the most part, still hidden or indistinguishable in that limbo which under the names of "undernutrition," "poor nutrition" or "malnutrition" is recognized by everybody when present in an individual child and to which not only dietary deficiencies but also illness, poor hygiene and even poor heredity may have contributed.

PRESENT DAY AMERICAN DIETS. Obviously our present American diets, whether for children or for adults, are not deficient in carbohydrates, and except among the very poor or ignorant only rarely in fruits that furnish vitamin C, so that for practical therapeutic purposes one needs chiefly to keep in mind the possibility of protein, mineral and fat-soluble vitamin deficiencies. For the most prompt correction of these (and time is an important factor when these occur during the period of tooth development) a high protein diet, in which liver, kidneys and eggs are prominently featured, extra calcium and phosphorus, cheaply and easily supplied as di-basic calcium phosphate, as mentioned in a previous paragraph, together with cod-liver oil to facilitate its utilization, are reasonable recommendations, soundly based, easy to carry out and requiring a minimum of expert oversight. But important as are cod-liver oil, orange juice, and even possibly a dash of copper with our ration of iron, in themselves they supply but an infinitesimal amount of the body substance, and the building of sound bodies and sound teeth must apparently rest on the broader base of abundant biologically high-grade foods coupled with good hygiene rather than on the addition to a poorly chosen diet of the particular adjuvants (vitamins or minerals) that cure or prevent rickets, scurvy, simple goitre or anemia.

ABNORMAL CONDITIONS OF DUCTLESS GLANDS.

The effects of abnormal conditions of the ductless glands, despite their relative infrequency, present problems of much interest to dentist and pediatrician alike. But in the field of endocrinology even more than in the field of nutrition there are still many uncharted wildernesses in which theory runs riot although keenly devised experiments have cleared and are clearing away the underbrush. And of so specialized a nature are these problems and of such relatively rare occurrence as compared with those of health, hygiene and nutrition, that the discussion of them at any length may more properly be left to endocrinological monographs than entered upon here. A few brief paragraphs will, however, sketch their broad outlines and serve as a basis for evaluating their possible relation to dental conditions in the individual child.

THE LIVER exceeds all other glands in size and in multiplicity of functions. Its functions in connection with the metabolism of protein, carbohydrates and fats give it vital importance in nutrition, while its detoxicating function places it in the first line of defense against chemical, bacterial and food poisonings. Toxic and nutritional damage, in which the teeth and supporting tissues may share, characterize, therefore, diseases of the liver which at every age, except in the case of acute catarrhal jaundice, carry a grave prognosis. Aside from Biliary or Hanot's cirrhosis in which deformities of the nails and thickening of the bones about the wrists

and ankle joints may occur, no *characteristic* effects on bones or teeth have, so far as is known to the writer, been described.

THE PANCREAS, however, has important relations to dental diseases, for the teeth of untreated diabetic children are susceptible to a type of caries distinguished by extensive and rapid progression and which may be promptly arrested by dietary treatment and administration of insulin. But the arrest of the caries does not end the diabetic child's dental troubles for it is often accompanied by calcareous deposits that cause a serious irritative gingivitis. Few, if any, childhood diseases, therefore, require more continuous dental oversight and care than does diabetes.

BALANCED FUNCTIONING OF ENDOCRINES. With growth, with the structural form of body parts and with calcification, the liver and pancreas are apparently little if any concerned. On the other hand, general body growth, its rate, its orderly and balanced progression, the arrival of maturity and, to some extent, the aging of tissues, is controlled in large measure and kept to normal pattern by the balanced functioning of thyroid, pituitary, sex glands, adrenals and, possibly, thymus.

DISORDERED FUNCTIONING of these glands may give rise not only to disturbances of general growth and skeletal development but also produce characteristic alterations in the eruption times of the two dentitions, in the spacing of the teeth and the shape of the jaws, and in the development of the roots or of the whole teeth. The relation of the thyroid to the formation of the centers of ossification of long bones, the characteristic teeth of cretins, the relation of the parathyroids to calcification, of the pituitary to the growth in size and length of bones laid down in cartilage, and the determination of male or female type of skeleton by the sexual glands, have been demonstrated beyond doubt or cavil. Even in such familial conditions as cleido-cranial dystosis (defective ossification of cranial bones and the clavicles), with its remarkable dental anomalies, the fact that it appears in successive generations by no means excludes the possibility of endocrine pathology since the skeletal and dental changes may be merely the expression and results of inherited endocrine anomalies which produce disturbance of their functions.

ENDOCRINOPATHIES. In the diseases of this group, as in the case of nutritional deficiencies, there are in addition to the characteristic changes by which they are recognized, profound effects on cell quality and vital resistance with, as in the case of cretins, increased vulnerability to infections. Needless to say, one should be cautious indeed about ascribing such general disturbances of health to endocrine causes unless there is unmistakable evidence of their presence.

INCIDENCE OF NUTRITIONAL DAMAGE HIGH; VARIATIONS IN STRUCTURAL FORM RARE. Such then, in brief, are the types of diseases (infectious, nutritional or endocrine in origin) that by reason of their specific nature or their indirect effects most commonly inflict damage on the growing organism. Nutritional damage (damage to quality and vital resistance of tissue) results from all diseases, while interference with structural form and size is limited to but few. Malnutrition and undue susceptibility to disease is very frequent, while anomalies in structure and development are relatively rare.

Finally, all diseases are capable of initiating those vicious circles in which children are poorly nourished because of such illnesses, and by reason of their malnutrition are in turn more vulnerable to further disease.

HISTORY OF CHILDHOOD HEALTH OF IMPORTANCE TO THE DENTIST. The dentist who has elicited a history, especially in the first years of childhood, of endocrinopathy, of long and serious illness, of definite malnutrition, or merely that the child has always been a "poor eater," has, it would seem, good warrant for assuming a poorer calcification and a lower resistance of teeth and their supporting structures to injury, disease and, even, to the stresses and strains of orthodontia, as well as the existence of less favorable mouth and salivary conditions than obtain among vigorous, well-nourished children who have escaped these hazards.

NUTRITION AND DIET

The number and something of the character and function of the nutritional elements essential for body building have been briefly discussed in the preceding paragraphs that dealt with the deficiency diseases. And for further details, if the reader desires such, regarding the chemical nature and biological functions of these dietary essentials, as well as their quantitative occurrence in various foods, reference may be made to the elementary textbooks of nutrition. Fortunately, Medicine, Dentistry and Nutritional Science are in complete accord as to the necessity of having all of these elements supplied by the diet and the present confusion in the field of dietetics is not as to what diet shall furnish *in terms of chemistry* but as to how this shall be done *in terms of food*. For we do not supply our tables with these essential elements purchased separately as such, but with the bewilderingly complex combinations of them as they exist in meats, cereals, vegetables, fruits and dairy products.

PHYSIOLOGY AND PSYCHOLOGY OF EATING.

This lack of agreement in the field of dietetics as to how scientific nutritional knowledge shall be applied to every-day eating

is, however, due not only to the difficulties of planning meals in terms of their constituents but even more to the wide gaps in our knowledge of the basic physiological and psychological phenomena inherent in human eating, and the fact that such data as exist are recorded chiefly in the literature of physiology and psychology and only in rare instances are to be found in textbooks on nutrition or dietetics. For these reasons it seems pertinent at this point to indicate the sources of such data used in the preparation of this chapter as are not readily accessible in dietetic literature and especially the data on which are based those statements and paragraphs which are not wholly in accord with current practice in child feeding.

AUTHOR'S DATA. Data of this sort have been largely derived from (1) twenty years of experience in feeding children by the usual pediatric methods; (2) five and one-half years of study of the natural eating habits and behaviour of children from weaning on, during the self-selection of diet experiment; (3) observation of children's eating in the orthopedic, cardiac and convalescent wards of the Children's Memorial Hospital in Chicago where a simplified application of the principle of self-selection of food has been used for the past five years. For the sake of clarity these two latter sources will be described later in more detail.

DIET LIST WHICH MEETS NUTRITIONAL REQUIREMENTS IS NOT SUFFICIENT. To feed children (or, for that matter, any group) successfully, requires something more than a knowledge of their nutritional requirements and a diet list that will supply these. The reasons are not far to seek: *eating* and *digestion* must precede the supplying of proteins, carbohydrates, minerals, etc., to meet the body's needs and eating and digestion impose their own *prior* requirements, psychological and physiological, upon the selection of foods; requirements that are set in the one case by the sensory, emotional and intellectual nature and, in the other, by the structure and functioning of the gastro-intestinal canal from mouth to anus and of the nervous connections of its various parts with each other and with the higher nerve centers. Furthermore, both sets of requirements bear the stamp of the human as distinguished from all other animal patterns.

Let us look a little more closely at these physiological and psychological requirements which must first be satisfied and see how and by what means they permit the selection or compel the rejection of various foods.

That it is the structure and functioning of the gastro-intestinal canal which make it impossible for man to exist as a grazing animal is, of course, a matter of common knowledge. But how and to what extent do they determine what of ordinary human diet can be fed to children?

DEVELOPMENT OF CIRCULATORY, RESPIRATORY AND GASTRO-INTESTINAL SYSTEMS. The circulatory and respiratory systems are mature in all but size at birth, that is, they proceed at once to function in essentially the adult manner. The gastro-intestinal system with its accessory glands, from lips to anus, is also well along the road to maturity though not equally so in all its parts, that portion from lips to stomach having yet to undergo considerable development before functioning in the adult manner. Lack of teeth for fine division of food and undeveloped coordination of the neuromuscular apparatus of the jaws, tongue and pharynx — indispensable for handling a bolus of food in the mouth and swallowing it without choking — necessitate a liquid diet. Digestion, too, in the first six months is not of the adult type, being chiefly intestinal since the stomach at this early age contains no free hydrochloric acid and the acidity of its contents is relatively low. Yet so rapid is the development of this upper part of the gastro-intestinal tract from lips to pylorus that by the last quarter of the first year several teeth have been cut, masticatory movements of the jaws have appeared, food, if finely divided, can be adequately mixed with saliva, worked into a bolus and swallowed and stomach acidity has increased to the point where free acid is present. In other words, the gastro-intestinal canal has at the age of nine to twelve months reached maturity so far as functioning in the adult manner is concerned, except in respect to the number and character of the teeth.

The implications of this early maturity of the gastro-intestinal tract and functions would seem to be that at the customary weaning age (nine to twelve months) the child can eat, digest and, therefore, use the common simple foods of adult life *provided only* that they are finely enough divided to prevent sticking in the small throat or, in the case of vegetables and fruits, to break up the cellulose which is not only rough and irritating but is wholly unaffected by either gastric or intestinal juices. That such is actually the fact can and has been proved by experiment as will be detailed in another connection.

HUNGER, APPETITE AND PLEASURE OF SATISFYING THESE DOMINATE CHILD'S EATING. The psychological factors naturally dominant in the young child's eating are hunger, appetite, the pleasure that comes from the satisfaction of these and the general sense of well-being and comfort that arrives with "enough" and temporarily ends all desire for food. This sequence, recurring day in and day out in its definitely established order is an intrinsic part of the nutritional mechanism, initiating and carrying on as it does the business of eating, and is in full working order at birth. Only when a child gets normally hungry by mealtime, eats with keen appetite and enjoyment of his food and ends his meals with the expansive feeling of good nature and well-being that comes from complete physical satisfaction, do we say that he is a "good eater."

These psychological requirements of hunger, appetite and their satisfaction are the hurdles over which the well-meant attempts of pediatricians to impose a "planned economy" on children's eating by the use of routine diet lists have stumbled. Why is this and what is their nature and their relation to digestion and nutrition?

HUNGER. The physiological mechanism of hunger has been thoroughly investigated and is well understood. It is centered in the stomach, which is the seat of mildly painful contractions when empty and is not under the control of the higher brain centers. Hunger rises into consciousness as an insistent demand for food, liked food if that can be had, if not, something, anything, to put an end to the discomfort. It initiates eating but is itself short-lived, disappearing as soon as a few mouthfuls have been eaten, when the stomach, no longer empty, ceases to have painful contractions. From this point appetite and the pleasure of its satisfaction carry on, the end point of eating being not the satisfaction of hunger but the satisfaction of appetite and the feeling of "enough."

APPETITE. But of appetite neither the physiological mechanism nor the natural behaviour patterns are so well known. The sensations by which it is immediately and largely, if not wholly, guided arise in the mouth and nose, while its physiological connections with the stomach make it the initial stimulus to the flow of gastric juice, as well as making the mouth "water." As long ago as 1905, Pavlov², the great Russian physiologist, demonstrated that in dogs *appetizing* food induced a greater secretion of gastric juice than did foods not so well liked by the animals, and Miller, Bergheim, Reh fuss and Hawk³ in 1920 reported like results from the investigation of the "appetite" or "psychic" secretion of gastric juice in normal men. On the other hand, its relation to the higher brain centers is sufficiently close to make appetite, though little susceptible to enhancement by intellectual or emotional means, highly so to depression even to the point of suppression, as seen in the loss of appetite accompanying grief, anger, anxiety or, even, mental concentration on absorbing work.

FOOD "ENOUGH." So much we can say with assurance. But we do not yet know what determines the end point of "enough" of all food, or the end points for each of the different foods of a meal, which latter are obviously not coincidental, since a child may have eaten all it wants of meat and vegetables and still have appetite for milk or fruit. We are ignorant, too, of how hunger and appetite are related to each other and, which is still more important, of how and to what extent desires for foods (appetites) of

² Pavlov, *The Work of the Digestive Glands*, 2nd English Ed. 1910.

³ Miller, R. J., Bergheim, O., Reh fuss, M. E., Hawk, P. B.: Gastric Response to Foods. *Am. J. of Physiol.* 52:1 (May, 1920).

different sorts are representations in consciousness of the body's metabolic needs.

BASIC METABOLISM NEEDS ARE PROBABLY REFLECTED IN CONSCIOUSNESS. Much circumstantial evidence both negative and positive in character points to the probability that basic metabolic needs are, at least to some extent, reflected in consciousness as appetites or cravings for the foods that will supply them and that are at the same time adapted to the gastro-intestinal structure and functioning of the species. Herbivorous animals will travel astonishing distances to a salt lick. Canibalism in chickens, in rats and in other animals is seen when diets are deficient in protein, certain minerals and vitamins; the newly weaned human infant, in the preparation of whose food no salt is added to vegetables and cereals, will eat plain salt, though crying lustily the while from the smarting it causes in his tender mouth; and children with rickets have been known to eat egg shells and plaster picked from walls for the calcium salts.

Conversely, men have neither appetite nor longing for the forage of the herbivores, nor do the latter evince desires for flesh — appetite thus according with gastro-intestinal structure and functioning.

APPETITE GONE AMUCK. Yet, on the other hand, we are daily confronted with examples of appetite gone amuck: children stuffing themselves with sweets or eating green apples with painful results, or showing no inclination to eat heartily of good, wholesome foods, although plainly poorly nourished. With such conflicting evidence and so many gaps in our knowledge of the physiological relations of appetite, it is small wonder that we are uncertain about its trustworthiness as a guide in the selection of either articles or quantities of food. Only more light on this subject can extricate us from the dilemma of trying to make children eat what we think good for them whether they like it or not, or letting them have anything and everything they please.

SELF-SELECTION FEEDING EXPERIMENT.

APPETITE AS A VALID GUIDE to the selection of food when only fresh natural foods are used and these are prepared by only the simplest ways of cooking, is presented by evidence of three sorts, viz., that of the survival of the human race from the earliest times under its guidance, unaided until recently by any scientific knowledge of nutrition; that derived from study of the diets and physical condition of primitive peoples; and that brought to light by direct experiment, i.e., allowing newly weaned infants to select their foods in the quantities they wished from a long list of fresh natural foods (meats, glandular organs, cereals, vegetables, fruits, eggs,

milk and salt), raw or simply cooked without loss of soluble salts and without mixture or added seasonings.⁴

PRIMITIVE PEOPLES. The vigor and excellent nutritional condition of many primitive peoples whose eating has been governed by the dictates of hunger and appetite is well attested by competent observers. Their "natural" diets, though varying widely in different parts of the world have been shown, when analysed in terms of food elements, to cover all the known nutritional needs and have therefore been relatively fool-proof. The precise experiment with the newly-weaned infants under conditions that precluded the handing down of dietary information or experience by either instruction or example has shown that on such fresh and simple foods as were used young children, with no other guide than hunger and appetite, thrive with apparently optimal nutrition and digestion.

THE SELF-SELECTION OF DIET STUDY⁵ REVERSED THE USUAL PRACTICE of telling children what and how much they must eat, and let them show us what and how much they would eat when undirected and uninterfered with. Fifteen children from six to eleven months of age who had not had foods other than milk were thus observed for periods varying in individuals from six months to four and one-half years. A large assortment⁴ of cooked and raw, uncombined, unseasoned and unsophisticated foods, consisting of food-stuffs commonly used by older children and adults, was served according to an unvarying schedule at three meals a day and no food given between meals. Servings were weighed or measured quantities (each food in a separate dish) and the remains were weighed or measured so that a complete record exists of the amount of each article eaten by every child at each meal. In the dining room each child had his own small table and all the foods for the meal were on his tray when it was set before him. Children ate with spoon or fingers as they could or wished and empty dishes were promptly refilled to ensure their having all they wanted of everything. The attendants were trained to refrain from conversation with the children during meals and never to comment on or show interest in what or how the children ate, their function being merely to wait on them and to help the youngest who could not wholly feed themselves.

Daily records of the children's weights, stools, and temperatures, were kept together with notes of anything unusual in their conditions; bi-weekly urine examinations and monthly blood counts

⁴ Meats: Beef, chicken, lamb, kidneys, liver, sweetbreads, brains. Fish: Haddock. Eggs. Milk: sweet and whole lactic. Cereals (all whole-grain): cracked wheat, oatmeal, yellow cornmeal, barley, rye. Vegetables: potatoes, carrots, beets, peas, cauliflower, cabbage, lettuce, spinach and turnips. Fruits: Apples, oranges, bananas, tomatoes, peaches, pineapple. Salt (sea salt).

⁵ Davis, C. M., Self-selection of Diet by Newly Weaned Infants. *Am. J. of Dis. of Child.* 36:651 (Oct., 1928).

and physical examinations were made, and Roentgenograms taken of their bones at frequent intervals. Anorexia, other than as a transient loss of appetite with acute parenteral infection, was unknown among them.

All of the children ate heartily with keen appetite day in, day out. It was not unusual for children to eat three or four bananas at a meal or five to seven servings of potatoes or meat. One child a year old frequently drank a quart of milk at his noon meal besides eating other food. Yet none were fat and all were solid, well-built children, healthy and vigorous, presenting no problems in behaviour. A few instances of vomiting or diarrhoea occurred with an acute parenteral infection but otherwise there were no digestive disturbances and never any constipation; laxatives were not needed for any of the group at any time during the period of the experiment. The study demonstrated that under the conditions of the experiment and with such natural foodstuffs young children could choose their diets and thrive without adult direction of eating, and the analysis of their self-chosen diets showed the soundness of their selection in terms of calories, proteins, fats and carbohydrates.

FOODS SERVED AT CHICAGO CHILDREN'S MEMORIAL HOSPITAL. In the simplified application of this way of feeding to meals in the orthopedic, cardiac and convalescent wards of the hospital⁶, no such array of foods is offered, but three hearty meals a day are served with more meats, fruits and common foods of every-day home life included than is usually the case in hospitals. Wienerwursts (a general favorite), pork chops, Hamburger steak, eggs, hardboiled, scrambled or fried, and pancakes find a place on the menus, and desserts are chiefly fruits, ice-cream or, more rarely, a pudding or pie. The food in large containers is put on the dressing cart and wheeled into the middle of the ward and the children choose what they want and have as many helpings as they like. Breakfast is hardly over before they begin to wonder what they are going to have for dinner and, except when fever or gravity of condition prevents, they eat with hearty appetites. Not only do they eat more than formerly but mealtimes have acquired a gay and festive air almost like a picnic, with children calling out what they want as soon as the cart is wheeled in. To the surprise of all, food waste has been sharply cut down, apparently because what a child chooses for himself he generally eats.

TRUSTWORTHINESS OF APPETITE AS A GUIDE. What then are the limitations to the trustworthiness of appetite as a guide in eating? First of all, it fails to protect against the ingestion of

⁶ Davis, C. M., A Practical Application of Some Lessons of the Self-selection Diet Study to the Feeding of Hospital Children. *Am. J. of Dis. of Child.* 46:743 (Nov., 1933).

unfamiliar poisonous plants and animals. Throughout the ages not only children but adults as well have eaten poisonous fish and mussels, mushrooms, castor-oil beans, and the berries of the deadly night shade. The explanation is apparently simple and not incompatible with the general validity of appetite as a guide, viz., that sampling is the primary method by which in omnivorous man appetite gains its information as to what is good to taste and satisfying in its results and, since sampling in the cases above mentioned may prove fatal, only tribal lore can protect against such "trick" varieties in which more often than not, either the appearance or the predominant flavor closely resembles that of edible varieties or is determined by nonpoisonous components. Appetite also fails as a guide and dietary deficiencies and digestive disturbances hitherto unknown appear among primitive peoples (as, for example, happened in Labrador and in the Faroe Islands) when highly milled cereals, sugar and canned goods replace the fresh, natural foods to which they have been accustomed. The fallibility of appetite in the new situation may be wholly real, i.e., due to the fact that the dominant flavors of the natural foods from which these more or less denatured products are derived to such a degree that appetite makes no distinction between them, or it may be that the verdicts of taste and smell are over-ridden by considerations of novelty, ease of preparation and the lessened labor by which daily fare can be procured. An experiment in which both fresh natural foods and also such derived and preserved products were served to newly-weaned infants for their free choice might throw light on this question.

SOME NATURAL FOODS HAVE SHARP END-POINTS FOR APPETITE; MOST REFINED FOODS DO NOT. Finally, there is some evidence that some natural foods have sharp end-points for appetite which are lost in large measure by the products derived from them. Thus, for example, in the case of natural sweets, honey, maple syrup and unrefined molasses, the end-point of appetite is sharp, coming suddenly after no great quantity is eaten. Just so much is wanted and no more. But with refined sugar products, candies, etc., one can go on nibbling throughout the day, unconscious of any sharp end-point of desire. The same is true of cereals. The end-point of desire for whole grain products is sharp but white flour crackers or bread can be nibbled away at until the desire for food of any sort whatever is gone. This absence of a sharp end-point of appetite, seen also in another carbohydrate derivative, alcohol, makes them habit forming in the sense that more and more can be taken at a given time without arousing the sensation of "enough."

Such in brief is the nature of the evidence we have at present on the validity and limitations of appetite as a guide to eating. It should, however, be noted before proceeding to the development and behaviour patterns of appetite in young children that in the

last analysis these fallibilities of appetite are the price of the amazing omnivorousness of the human race.

OMNIVOROUSNESS. The implications of omnivorousness are as important for dietetics as for biology and history. For ability to exist on wholly carnivorous diets or (with more difficulty) on diets exclusively herbivorous, and to satisfy nutritional requirements with the flora and fauna of every climate from the arctic to the equator is not only the basis of man's freedom to range but is also of the greatest survival value. So profound and far-reaching, indeed, are the effects of omnivorousness on appetite development and behaviour in the selection of diet that in the language of present day psychology these may be said to be conditioned by it. Its relation to the interesting question of an "optimal" diet for human beings will be at once apparent if we consider two examples where Nature has arranged an optimal diet for mammals. There still survives in Australia, though in rapidly dwindling numbers, an example of a class of mammals once fairly numerous, viz., the Australian Teddy-bear or Koala (*Phascolarctus-cinereus*). So perfect is the adjustment of its dietary habits and appetite to its nutritional needs that there is no room for choice in the matter of its foods. For, except when driven by extreme hunger, this Koala bear will eat nothing but the leaves of a certain variety of *Eucalyptus* tree with occasional bits of bark or *Sphagnum* Moss, the stems of which are filled with water (they drink no water), and if, driven by hunger, it eats aught else than this truly "optimal" diet, it sickens and dies. Its range is sharply circumscribed to that of its food and the perfection of dietary adjustment may prove its doom.

NURSED VERSUS ARTIFICIALLY FED BABIES. This perfect adjustment to one optimal diet in terms of food also characterizes the young of all mammalia during the nursing period. True, there has been evolved by careful study of this optimal food, woman's milk, and modifications of other milks, foods that will more or less adequately nourish infants and permit of good development, but we still do not know that even the most blooming artificially-fed infant is the equal, nutritionally speaking, of the breast-fed one, or whether he develops into as rugged an individual with as high a vital resistance of tissues. The surprisingly greater death rate reported by Grulee, et al⁷ among the artificially-fed infants under the care of the Infant Welfare in Chicago as compared with those that were nursed, certainly leaves the question still open.

After the weaning period, instead of living on a simple, optimal diet of one food, the infant lives on diets better or worse. Precision of adjustment gives way to multiplicity of possibilities

⁷ Grulee, C. G.; Sanford, H. N., and Herron, P. H.: Breast and Artificial Feeding, *J. A. M. A.* 103:735-738 (Sept. 8), 1934.

and perfection in nutrition to a greater chance of mere survival. Choice has entered, bringing with it the possibility of mistakes, and appetite has ceased to be completely infallible as we have seen in a previous paragraph. From fresh, natural, unprocessed foods simply prepared, however, appetite is still competent to select a sound diet, though it does not inexorably point to a certain one or few foods as in the case of animals.

The mechanism by which this is done is still unknown, but the manner in which food tastes and appetite behaviour develop in the young child at weaning time was beautifully demonstrated by the infants on the self-selection of diet experiment.

DEVELOPMENT OF FOOD TASTES AND APPETITE BEHAVIOR. Since appetite is the desire for foods that please by tasting or smelling good to an individual, it would seem that in the absence of such sensory information, i.e., if one has never tasted or smelled a particular food, he can neither really like nor dislike it. That this is actually true was shown by these infants. At first their choices were quite evidently wholly random; they would try not only foods but would hopefully chew the spoon, the dishes or the edge of the tray. All the articles of food on the long list were tried by every child and that not once only but several times, their faces showing surprise, followed by pleasure, indifference or disgust. But, after the first few days, they began to reach eagerly at once for some foods and to wholly neglect others so that their likes and dislikes were clearly apparent. And never again did any child eat so wide a variety of the foods served as in that initial period of sampling.

Tastes and dietary habits then develop on the basis of sampling, that is, by trial and error, and are reinforced by the satisfaction and comfortable feelings arising from the ingestion of certain foods and by habitual use of them, as well as by the natural preference for the familiar over the strange that is a part of childhood mental make-up.

The pattern of tastes in food as in all other things where choice enters in, is individual in character, the likes of children having as much in common and as much individual variation in foods as in clothes or in play, and whether individual children like few or many foods is also an individual matter, wideness or narrowness of range of tastes in eating seeming often to be paralleled by a similarly wide or narrow range of likings in other fields. In this respect children in general show three rather well-marked types.

Those, who from weaning on, like and eat almost everything, even their first olive and oyster being exclaimed over with joy.

Those whose likings are at first limited to a moderate number of the more commonly liked fruits, meats, vegetables and cereals,

but who as they grow older come to like more and more foods, quite naturally and without being urged to eat them.

Those who, as children, like only a strictly limited number of foods (which, however, cover all their nutritional needs) but who, as they grow up, learn to eat others as a matter of social conformity or convenience.

Many of the children who steadfastly refuse all vegetables are in this latter class.

DIET REGIMENTATION HAS TAKEN APPETITE AWAY. So lengthy a discussion of appetite may seem out of place in a chapter of this sort, but the experience of pediatricians and of parents in the last twenty years in trying to make children eat what we thought they ought to, regardless of appetite, has demonstrated that appetite cannot be coerced. In the oft-quoted words of Dr. Lippman of St. Louis, "With all our weighing and measuring, and all our rules and regulations as to when, where, what and how much to feed children, we have succeeded in doing just one thing — we have taken their appetites away."

If we cannot coerce appetite, and the nutritional requirements are fixed, how are we going to manage children's feeding so that they will be properly nourished? That is the practical problem of today in which parents, dentists, physicians and nutritionists are rightly concerned and to which our attention will now be turned.

FOODS WHICH SUPPLY NUTRITIONAL REQUIREMENTS

Fortunately, the science of nutrition has not only given us a clear picture of human nutritional requirements but has also determined what the foods are that provide these essentials most abundantly and which are, therefore, of the greatest biological value. These are: animal protein foods, among which liver, kidneys and eggs rank highest since they have all of the needed amino-acids which muscle cuts of meat do not and also because they are rich in phosphorus; fresh fruits and vegetables, the latter when cooked in such a manner as to prevent loss (by leeching) of mineral salts and other soluble products; wholegrain cereals; yellow animal fats (cream, butter and beef fat), which rate much higher than the vegetable oils and are the only carriers of the fat-soluble vitamins; and milk. Fortunately, again, such are the foods that are in general best liked by children, especially when prepared in the most simple ways, separately rather than in mixed dishes of which children like but few, without sauces or dressings, and with little seasoning. From these foods of highest nutritional value the meals of growing children should be in the main provided, since such foods provide a diet not only more fool-proof from the point of view of possible deficiencies, but it is with such foods that appetite has been shown to be a competent guide both for the eating of primitive peoples and young children. In the absence of more precise information than we now have it would seem that the rigid and

absolute exclusion of all processed foods and those of inferior biological value is unnecessary, but that they should be relegated to a distinctly minor place in the child's dietary.

SUGAR. But of one such product, sugar, there is a growing distrust both among nutritionists and dentists, and particularly of its being eaten between meals in the form of candy, etc. So far as the author knows, no valid evidence has as yet appeared that sugar in moderately large quantities is not well tolerated by children or that it does them any nutritional damage per se. The fact remains, however, that sugar is a source of "quick calories," a prompt quieter of hunger and it seems unlikely that when a large proportion of the child's daily calories are furnished by sugar he will have appetite for, or actually can within his caloric limits, eat enough of nutritionally necessary foods to ensure the best development and quality of tissues and organs. In the experience of the author those children in whom the use of sugar has been sharply limited, i.e., no sugar on cereals or other foods, and only rarely a piece of candy, have been larger eaters, with better teeth, than those who regularly consumed sweets in quantity.

When once meals consisting chiefly of the highest grade foods have been provided for children, they will eat more and fare best if left to eat what and as much as they want of them, without suggestion, urging, nagging or comment, that is, if they are allowed to eat them as adults do, without direction. This results, of course, in astonishingly large meals being eaten at times and, at times, very small ones, but such are apparently automatic adjustments made by appetite to periods of unusual activity, extremes of temperature, etc.

HOW MUCH FOOD FOR CHILDREN? Important as is the selection of proper foods, the subject of diet in relation to the nutrition of children is not wholly covered by the discussion of *what* they may have to eat and what are the foods of highest biological value. There still remain the equally important questions: How much food should children have, and what are the basic considerations which should guide the handling of this phase of diet?

STANDARD DAILY REQUIREMENT IMPRACTICAL. Here, as will presently be seen, we must bid farewell once and for all to the idea of carrying over from the field of medicine to that of diet, the practice of accurately measured daily dosage. In feeding we deal not with materials alien to the body's composition in health, where a fine line divides the therapeutic from the harmful dose, but with body-building materials in the supplying of which not only must all known requirements be covered, but provision be made for a large margin of safety, as Nature herself has done. How large are the factors of safety she has used in designing the body is quickly apparent when we recall that human beings can carry on with one lung, or one kidney instead of two, or with slightly less than fifty

per cent. of sound liver tissue, provided only that emergencies of hard labor, exposure or disease do not call for the no longer existing reserves.

Life, however, even under the most favorable conditions, is more or less a succession of such emergencies—stresses and strains, exhaustion, exposure, illness—that call for quick marshalling of reserves, and wisdom counsels the provision of wide margins of safety in diet and nutrition to meet them. We have no means of accurately estimating how wide this margin of safety should be (either in calories or in food elements—protein, fat, etc.). We can only say that its upper limit cannot be above what the healthy, well-nourished child with good appetite will eat with comfort and without urging. But even if, hoping for the best, we make no provision for this margin of safety that eludes our units of measurement, we shall not find that the beguilingly simple definition of the child's quantitative nutritional requirements, viz., "the amount of food needed for body maintenance, growth and activity," will be very helpful in feeding the individual child. The *precise determination* of the quantities that healthy, well children should eat, whether by age or by weight or on a daily basis is *wholly impracticable* and all but, if not quite, impossible. It is true that in the laboratory, by the use of the calorimeter, the amount of food used by a person at rest and, therefore, the amount required for maintenance of body weight at that time and under those conditions can be accurately calculated. But in growing children, body conformation and surface area are continually changing even the maintenance figures, while variations in activity and in the temperatures both in-doors and out to which they are subjected, give rise to such violent swings in the demands for food to supply heat and energy as to make *calculations of their needs, whether for maintenance or activity, practically valueless* from the point of view of setting up meals. It might seem at first blush that the difficulty could be solved by prescribing for children of varying ages the averages of the quantities determined calorimetrically to be necessary for body maintenance both at rest and under conditions of violent exertion, with a fixed percentage added for growth requirements and insisting that these quantities be eaten regularly day in, day out. Practical experience with a rough approximation to this method of setting quantities, i.e., the pediatric diet list with quantities of each article prescribed on an age and/or calorie basis, has shown that such attempts to adjust quantities to the child's needs are much like calculating the mean temperature for the year, having the carburetor set accordingly and expecting satisfactory operation and uniform gasoline consumption spring and fall, summer and winter. The provision of meals from such a diet list is easy; getting them eaten in exactly the quantities ordered, a major family trial, with anorexia a common sequel and the net results, digestive and nutritional, far from satisfactory.

Hunger and appetite are still the most accurate means for regulating the quantities that children should eat. Nor should the fact that we do not know by what mechanism the sensation of "enough" is produced in consciousness deter us from trusting its operation when plain, wholesome foods, simply prepared, are served, or tempt us into definitive limiting of the quantities children may eat when there is yet no sound basis on which to do so. In this connection the reader may be reminded that after many laboratory determinations of the nursing infant's caloric needs, the figures determined by weighing breast-fed infants who nursed to complete satisfaction were found to vary less and, as judged by results of use, to be the more accurate.

Wide variation in the size of meals eaten characterizes the natural pattern of eating behaviour as seen in wild animals and primitive man. Not only is there stuffing and starving as food is plenty or scarce, but living as these do under conditions in which the most violent physical exertion alternates irregularly and in no fixed proportion with indolence, meals are huge, moderate or small, accordingly.

DIET REQUIREMENTS FOR ADULTS. Civilization and machines have changed all this for adult men and women. Peaks and depressions in the curve of effort have been leveled off and the demand for physical exertion greatly reduced in every occupation. As a result, the food consumption of adults has declined on the average to a figure (measured in calories) nearer to a mere maintenance level than any ever before known. Under such conditions which resemble in many ways those of laboratory animals, it is difficult indeed for adults to realize either how great is the quantity of food that children need or how varied in size will naturally be the meals they eat. Yet children who, school life notwithstanding, still race and fight and play, putting forth their utmost efforts, *are more akin in their requirements and eating behaviour to animals and primitive man than to their modern parents.*

The "phantom" breakfast and the dainty luncheon that enable a mother, relieved of all heavy manual labour by the mechanical and electrical equipment of the home, to keep her girlish figure, and that satisfy a father at his office desk or tending a machine, have no place in the feeding of children. Indeed, such meals are in part responsible for the quantities of sweets children eat between meals. Farm type breakfasts with eggs or liver or kidneys, whole-grain cereals, milk and fruit, send a child off to school with food that will last and cover his nutritional needs, while equally hearty dinners and suppers will carry him through the hard play he should have after school and send him to bed at night with that well-filled feeling which is in itself conducive to prompt and sound sleep.

EXAMPLES OF QUANTITY OF FOOD EATEN BY CHILDREN. Some idea of the quantities that healthy, active schoolboys eat when they

can eat as much as they wish of plain, hearty meals may be seen from the following records of four consecutive days' meals eaten by two boys, eight and nine years old respectively, who were taken for the self-selection of diet experiment when seven and eight months old and since the closing of the experimental nursery in 1931 have been members of the writer's family.

BOY A. 8 YEARS, 9 MONTHS OLD

- 7:30 A.M. Orange juice; puffed rice; 4 strips of bacon; 2½ waffles with maple syrup; ½ glass milk.
- 12:30 P.M. Soup; celery, olives and raw carrots; 2 helpings of lamb; potatoes; stewed tomatoes; 2 helpings ice cream.
- 5:30 P.M. Shrimps; lettuce; 3 slices cold lamb; 2 slices bread; strawberries.
- 7:30 A.M. 4 large prunes; cereal; scrambled eggs; 1 glass of milk.
- 12:30 P.M. 1 large pork chop; 3 helpings potato; 2 slices bread; 2 glasses milk; ice cream with strawberries.
- 5:30 P.M. Very large helping minced lamb; 2 helpings canned corn; 1 roll; 2 glasses milk; 2 rolled pancakes.
- 7:30 A.M. 3½ shredded wheat biscuits with cream; 1 boiled egg.
- 12:30 P.M. 3 pieces of veal; potatoes; 1 baked apple with raisins.
- 5:30 P.M. 8 kidneys; 2 large helpings wild rice; 2 slices bread; 3 glasses milk; stewed apples.
- 7:30 A.M. Orange juice; 2 large helpings of oatmeal; 2 slices bacon; 1 glass milk.
- 12:30 P.M. 6 pieces veal; 4 boiled potatoes; 6 pieces ryekrisp; 2 butterballs; apple pie with cheese; 1 glass milk.
- 5:30 P.M. 6 potato cakes; large and frequent helpings raw carrots; 3 stalks celery; pear salad with cottage cheese; 2 glasses milk.

BOY D. 8 YEARS, 3 MONTHS OLD

- 7:30 A.M. Orange juice; puffed rice; 2 waffles with maple syrup; 4 strips of bacon; 1 glass milk.
- 12:30 P.M. Soup; lamb; potatoes; stewed tomatoes; rolls; celery, olives and raw carrots; ice cream.
- 5:30 P.M. Shrimps; lettuce; 2 slices cold lamb; strawberries; 2 slices bread; 1 glass milk.
- 7:30 A.M. 4 large prunes; cereal; scrambled eggs; 2 slices buttered toast.
- 12:30 P.M. 1 large pork chop; spinach; 2 helpings potatoes; 2 slices bread; 2 glasses milk; ice cream with strawberries.
- 5:30 P.M. Very large helping minced lamb; 2 helpings canned corn; 1 roll; 3 glasses milk; 1 rolled pancake.
- 7:30 A.M. 4 large prunes; 3 shredded wheat biscuits with cream; 1 boiled egg; 2 pieces buttered toast; 1 glass milk.
- 12:30 P.M. 2 pieces veal; potatoes; broccoli; 1 piece bread; 1 baked apple with raisins; 1 glass milk.
- 5:30 P.M. 4 kidneys; large helping wild rice; 2 slices bread; stewed apples; 1 glass milk.

- 7:30 A.M. Orange juice; oatmeal; scrambled eggs; 2 slices of bacon; 2 slices buttered toast; 1 glass milk.
- 12:30 P.M. 2 pieces veal; beans; 3 boiled potatoes; 2 pieces ryekrisp; apple pie with cheese; 2 glasses milk.
- 5:30 P.M. Large helping chipped beef; 4 potato cakes; 2 slices bread; pear salad with cottage cheese; raw carrots (very large helping); celery; 2 glasses milk.

(Note: "pieces of meat" mean pieces about 4 inches long, 2 inches wide and 1 to 1½ inches thick. Cream, not milk, served with cereals.)

CHILDREN WILL NOT OVEREAT. The fear that children will overeat if not stopped is a bogie often present in the mother's mind if she sees meat, potatoes, butter, eggs, or fruit disappearing from a child's plate in quantities far larger than she herself would wish or could eat. But few indeed are the children brought to the physician's office with complaints arising from over-eating, and that such a fear is wholly unfounded when foods are plain and simply prepared was fully demonstrated in the self-selection of diet experiment. Far more common and potent causes of digestive upsets in children exist and will be briefly discussed under the hygiene of eating habits. The greater danger lies in limitation of quantities and failure to provide that ample margin of safety above the requirements of growth, maintenance and activity which will from time to time be needed for unforeseen emergencies.

HYGIENE

Given health and good nutrition, the child attains that perfection of structure and quality of tissue which enable him, within the limits set by his heredity, to realize his utmost possibilities in vitality and vigor.

HYGIENE OF EATING, EXERCISE AND REST. But health and nutrition are in no small measure dependent on hygiene, which may be defined as a *healthful way of life*, for hygiene, or this healthful way of life, provides that combination of circumstances in which the child's activities, rest and eating habits function to his greatest advantage, while without this fortunate coordination neither health nor nutrition can be maintained at a high level. Adequate sleep, abundant exercise and good eating habits make up, then, the sum of the child's requirements in the field of personal hygiene.

CLEANLINESS, which to many mothers is the beginning and end of hygiene, is deliberately omitted from this list. Too many aboundingly healthy and sturdy, though appallingly dirty little rascals in town and country refute daily the idea that to be healthy a child must be clean, while the dental dictum that a clean tooth never decays has not stood the test of time. The importance of training children in personal cleanliness is not to be gainsaid, but it rests on other, if equally valid grounds, viz., the necessity of

forming habits that will make them acceptable members of society and that will deter them from spreading infection by hands contaminated with sputum, nasal discharges or other excreta.

EATING. The hygiene of eating habits demands the preservation of the natural sequence of hunger-appetite-satisfaction, without which children are rarely well-nourished, although sedentary adults may go for long periods eating regularly although seldom experiencing the pangs of hunger and apparently maintaining good health and nutrition. *To become hungry* children require not only adequate exercise, but also adequate rest, freedom from emotional strain and excitement at mealtimes, and intervals between meals long enough to allow the stomach to become completely emptied. After meals adequate in protein and fat the stomach is rarely empty under four or five hours time. *Three meals a day, therefore, from weaning age on, with nothing to eat between meals*, satisfy the last requirement of giving intervals long enough (as was demonstrated in the self-selection of diet experiment) for complete emptying of the stomach and the onset of real hunger without imposing undue delay in its satisfaction. "Hungry between meals" when adequate meals are supplied and eating is hearty is, thus, seen to be almost a physiological absurdity as can usually be demonstrated by offering a child a few crackers or a piece of bread, for real hunger does not deal in the nice distinctions of appetite but eagerly seizes whatever is offered. Frequent small meals, the mid-morning glass of milk and, above all, sweets between meals, actually lessen the total daily intake of food, and, in the case of sweets, cookies, bread-and-jam, etc., impoverish the diet in quality as compared with three "square" meals a day and nothing else.

ATMOSPHERE OF MEAL TIME. The three-meal-a-day schedule, however, presupposes not only adequate meals, but also the protection of mealtimes from untoward circumstances that depress hunger and appetite, interfere with the pleasure of eating and upset digestion. The hygiene of mealtimes requires a pleasant atmosphere, free from strain or hurry, and a rested, unexcited child. Battles between parents and child, whether on the subject of eating, deportment or school performance, are in the highest degree unhygienic at mealtimes, for anger, grief, fright, anxiety or excitement of any kind, even pleasurable, not only promptly depress or banish appetite and hunger, but by reason of the connections of the brain and higher nerve centers with the gastro-intestinal tract, interfere with the secretion of gastric juices and with the orderly rhythmic action of gastro-intestinal musculature. The vomiting of fright and the diarrhoeas of students at examination time are familiar examples of the profound effects of such emotions. "Better a dinner of herbs where love is, than a stalled ox and hatred therewith" is the expression of a physiological and psychological verity. Neither play nor any entertainment should be provided at chil-

dren's meals. Nothing can equal the enjoyment and satisfaction of eating when hungry and the introduction of extraneous pleasures or ideas only divert and distract attention from the meal itself. Children under seven or eight years of age actually *eat more food when they eat by themselves* than when they have their meals with older members of the family where their attention is distracted by conversation addressed to them or in which they are tempted to take a part. Occasional exceptions to this are seen in families with only one child; nor does it hold true, curiously enough, when the old rule so often quoted as an example of the parental hard-heartedness in olden times is enforced: that "children at table should be seen and not heard," for once the possibility of taking part in a conversation and thus holding the center of the stage from time to time is removed, the ordinary conversation of adults has slight power either to divert or hold the attention of young children from their own occupations.

Even good meals and quiet pleasant mealtimes will fail, however, to accomplish our purpose—hearty eating and good digestion—unless the child comes to the table *rested, relaxed and with hunger uppermost in his mind*. Children seldom eat heartily, and are prone to digestive upsets if they do, when they are allowed to play ball, skate or go on with other strenuous and absorbing occupations until the meal is actually on the table and then sit down to eat, when they are hot, tired and disheveled, with muscles tense and minds absorbed in their recent occupations, knowing that the more quickly their food is bolted, the sooner they will get out to play. Calling children in from play twenty to thirty minutes before mealtimes, getting hands and faces washed and hair neatly combed and then providing them with toys or books for the remaining minutes, allows cooling off, relaxing of mental, emotional and physical tension, and adequate rest. Hurrying through meals is also easily prevented by removing the object for so doing.

CHILDREN'S DIGESTIVE UPSETS are commonly laid by mothers to the eating of some particular food or to eating "too much," with little consideration to the circumstances under which the meal was eaten. Yet, aside from infections, other than through the alimentary canal, no other cause of digestive upsets has been so common in the experience of the writer as violations of these elementary rules of the hygiene of eating and eating habits. Even the much maligned circus lemonade would probably be quite harmless were it not for the excitement and fatigue of a long afternoon under the "big top."

REST AND SLEEP. So profound are the effects of fatigue and particularly of chronic fatigue on children's appetites, eating and digestion and, hence, on their nutritional state, that we are brought quite naturally to a consideration of the hygiene of rest and sleep—a subject in which dentists have a peculiar and especial interest

because of the possible bearing of sleeping positions on the occurrence of deformities of the jaws and malocclusion.

We are accustomed to say that children should sleep thus and so many hours per night and, if we substitute for the word "sleep" the word "rest" we are quite right. The *sleep* requirements of children vary as much in different individuals and from day to day in the same individual as do food requirements; as was shown by studies of the natural sleeping habits of children made at Vassar College in 1930 and 1931, periods of long night sleep are often followed by periods of shorter ones and, whether young children sleep at nap time depends much on their activities and on the length of night sleep as well. Such being the case we cannot reasonably demand that a child should sleep the same number of hours every night. We cannot force him to do so, and so far from congratulating ourselves if he does, should rather suspect that we are not giving him the opportunity for as many hours of sleep as he could often use. In the handling of this question, then, common sense indicates (as in the matter of food) the provision of a wide margin of safety in the hours assigned for rest that will always cover his maximum requirements whether he sleeps or not.

THE EARLY AFTERNOON REST, so frequently abandoned after the first three years, may be advantageously continued up to school age and on holidays and during vacations until the age of nine or ten. Children differ less from adults in capacity for intense activity than for sustained and continuous effort; their strength is no measure of their endurance. Naturally, after the age of three or four years one should not expect children, who have properly early bedtimes, to sleep during the afternoon rest although they may sometimes do so, but resting quietly and alone in a darkened room permits that relaxation of mind and body, that freedom from demands on attention, that cessation of the impingement of a complex world and of other personalities on an immature nervous system which are only less valuable for rest and recuperation than sleep itself. From school age on, books or simple puzzles give occupation for the rest period not too engrossing to be quickly dropped if the child becomes sleepy.

Chronic fatigue lies at the bottom of many a poorly nourished child's troubles. The over-tired child has difficulty in getting to sleep at night and so is often, mistakenly, put to bed later in consequence. His sleep is light, he is easily aroused and, not infrequently, is disturbed by unpleasant dreams if not by true nightmares. He plays less vigorously and for this reason as well as because of the direct effect of fatigue on appetite eats less than he should and is often constipated or has minor digestive disturbances. Physically he is characterized by poor muscular tone and an increased nervous irritability that leads to actual loss of energy in "fidgeting" and purposeless activity and in emotional outbursts on slight occasion.

SLEEPING POSTURES. What influence sleeping positions and the kind of beds children sleep on have in the production of deformities of the jaws, malocclusion, round shoulders and scoliosis, is a controversial question which is approached by the writer, it must be confessed, with some trepidation, for what evidence there is, is highly conflicting.

There can be little doubt but that a bed placed with its head rather than its side to the wall, so that the child does not lie always on the same side, a firm elastic mattress, no pillow, and bedclothes loosely enough tucked in to avoid all pressure and constraint, afford the most favorable conditions for sleep in the position of full extension. This is the sleeping posture which infants naturally assume, the one in which the body is most symmetrical and in which there is the greatest relaxation, since postures of effort are chiefly those of muscle flexion and those of rest characterized by extension. Furthermore, the posterior flattening of the skull that occurs when infants in their early weeks are kept bound against a board by swaddling bands as is the custom among Syrians, is an example of deformity produced by pressure that cannot be gainsaid. Every doctor and dentist sees from time to time, children with deformities so closely corresponding to the pressure of a hand under the jaw or to pressure brought about by some other habitual peculiarity of the sleeping position that the relation seems indubitable.

On the other hand many animals habitually sleep "curled up" as do countless children who assume this and other unsymmetrical positions in adjustment to their miserable beds, with often a hand under the jaw to turn the face for freer breathing and yet show no deformities.

DEFORMITIES MAY BE DUE TO INSUFFICIENT CALCIFICATION. The reconciliation of such conflicting evidence would seem to lie (and here the author is but expressing her own opinion) in the probability that in individuals in whom calcification is deficient or delayed, muscles hypotonic, or ligaments, fasciae, and tendons, lacking in elasticity, whether as the result of prematurity of birth, endocrinopathy, malnutrition or serious illness, faulty sleeping positions may and do determine such deformities. But that such deformities can be produced solely by poor beds and unusual sleeping positions in vigorous, well-nourished children whose bodies possess much the same resiliency as healthy twigs which must be *continually* bent to be trained in directions contrary to their innate law of growth, seems to the writer not only unproven as yet, but on the whole unlikely.

There is still room for much improvement along the lines of more hygienic management of children's eating habits and hours of rest. But so rapidly has spread an appreciation of out-of-door life and the benefits of exercise since the turn of the century, with consequent development of playgrounds, etc., that the child hunched

up over his book with no interest in outdoor play has become a rare avis. Fortunately too, for exercise influences favorably not only muscular development and circulation, but also sleep, appetite and the nervous "tone" of the child, toughening and strengthening this as it does his muscles.

EXERCISE. In all of these respects the younger the child, the better are the results of free play in comparison with those of supervised and set exercises or athletics. There is an increasing tendency to postpone the age at which children should be permitted to engage in competitive athletics in any formal sense to one much nearer that of full bodily maturity than was the practice a few years ago. The duration and violence of exercise to be allowed children is to be judged in large measure by the amount of fatigue it induces, since in order not to defeat its own purpose, by overstraining heart muscle or interfering with appetite and sleep, it should not exceed that from which a *short* rest will give prompt recovery. Errors are more often made in the length of time children are allowed to pursue active sports without intervals of rest than in the violence of the sports permitted, since it is by lack of capacity for *sustained* and *continuous* exertion whether physical or mental that children differ most from adults in the field of effort. Only maturity and the presence of the reserves of strength and nutrition then acquired develop endurance.

The younger the child the more definitely should exercise and sports be *restricted to the daytime*, since the practice of letting children go out for hard play after supper, with the attendant excitement, is often responsible for difficulty in getting to sleep and for restless sleep when it does come.

EXERCISE OF THE MUSCLES OF MASTICATION is a subject that is seemingly worthy of more attention than it has received since the advocacy of excessive and measured mastication of each mouthful fell into disrepute. No animal has to be taught to chew or to use his jaws sufficiently to ensure proper development, yet not all young children have the opportunity they need for such exercise, partly on account of the condition of their teeth due to neglected decays, and partly due to the soft character of their food. The constantly prattling toddler not only acquires language thereby but also gives his jaws and tongue much exercise that the silent child and the one left for the greater part of his day alone misses. Children of weaning age, as was mentioned in a previous paragraph, are able to chew well after only a few days practice and do so with vim and gusto, unless the anxious mother or pediatrician insists upon sieving and pureeing of their foods, and/or adding milk or other liquids to their solid foods so that they can be easily swallowed without chewing and admixture of saliva. If these practices be avoided and food for small children be divided only so finely as to

ensure swallowing without choking, good chewing habits will develop naturally and there will be little bolting of foods that by nature require admixture with saliva. Furthermore, meat (the digestion of which is not aided by the admixture of saliva) may leave the stomach too soon, i.e., before it is thoroughly impregnated with hydrochloric acid, if it be too finely divided or scraped free of all connective tissue, and undergo putrefication in the intestine, thus causing diarrhoea.

RECIPROCAL ACTION OF HEALTH, NUTRITION AND HYGIENE.

A chapter covering so wide a field must of necessity be limited to a general view, thereby omitting a wealth of interesting and more or less important detail. If, however, the present chapter shall have reminded the reader of how ingrained are the natural behaviour patterns and the automatic regulations of complex mechanisms, physiological and psychological, that have been evolved in the development of the human child from the lower animals and of the extent to which the reciprocal action of health, nutrition and hygiene forbids concentration upon one of these to the exclusion of the others, the chapter will in some measure have accomplished both the purpose of the author and of the work of which it forms a part.

The Dentist's Interest in Health, Nutrition and Hygiene

The following comments on Dr. Davis' article are presented by the author:

Attention has been called to the fact that disease and malnutrition very rarely cause changes in gross structures of the body (of which the dystrophies of the teeth are excellent examples), while damage to the quality and vital resistance of tissues in general is almost universal. It has been pointed out, also, that disease and malnutrition are to a large extent inseparable; malnutrition may so effect the quality and vital resistance of cellular structures as to make the individual more vulnerable to disease, while disease so increases malnutrition as to indirectly cause serious cellular damage to all of the tissues, which may be more far reaching than the actual damage resulting directly from the disease.

Knowledge of this vicious cycle, and particularly the long continued effects of malnutrition after one has recovered from the disease, are of much value to the dentist in developing an understanding of susceptibility and immunity to dental caries. For example, in the case report in Volume III, page 247, there occurred a change from practically absolute caries immunity up to the thirty-eighth year, to high susceptibility as a result of typhoid fever,

and this susceptibility was continued over a period of two years after the patient had recovered from the illness. In this case, two years were required to repair the nutritional damage to the cellular structures after the patient had recovered from an attack of typhoid fever which lasted a few weeks. When the nutritional damage was fully repaired, immunity to dental caries was regained.

In view of the fact that there are definite age limitations to the occurrence of gross structural damage to the teeth and also because the most reliable information indicates that cellular or metabolic changes do not occur in the enamel after it is formed, it seems desirable to consider the possible effects of disease and malnutrition on susceptibility to dental caries, on the basis of periods of development and growth. In doing so, the question of caries susceptibility will be briefly discussed from two points of view: (1) the possible effects on the structure of the teeth, which might make the enamel more vulnerable to caries; (2) the possible changes in susceptibility to caries as a result of changes in the environment of the teeth, particularly in the saliva.

DURING PREGNANCY. Defects in the gross structure of the temporary teeth are of such rare occurrence as to justify the statement that dystrophies of these teeth do not occur as a result of disease or malnutrition of the mother during pregnancy. It is also generally recognized that the foetus participates in very minor degree in the cellular damage suffered by the mother. The "nutritional robbery" of the mother by the foetus seems to afford the foetus a high degree of protection from the vicissitudes of the mother during this period. Therefore, the dentist has no special interest in the health and nutrition of the mother beyond that of the physician, whose duty it is to see that the diet is such as to safeguard the health of the mother and the general requirements of the child. There are no nutritional requirements of particular import to the development of the teeth, which are not covered by those of the body as a whole.

DURING THE NURSING PERIOD. On the average, the formation of the first permanent molars begins approximately at birth, although there may be considerable variations. Calcification of the central incisors may make some progress during the nursing period and there may be a beginning of calcification of other permanent teeth within the first six months. However, this development is confined to the inner layers of the enamel as marked by the lines of Retzius and does not include the outer surface, where decay begins. The crowns of the temporary incisors and first molars are practically completed before birth, and those of the cuspids and second molars are considerably advanced during the nursing period. The dentist has a special interest, because of the fact that hypoplastic defects of both the first molars and the incisors result

from periods of malnutrition during the first year of life, also because of the far reaching effects of cellular damage from lack of proper nutrition during the nursing period, as evidenced by Gru-lee's statistics which show a considerably higher death rate among artificially fed babies. However, there remains the question as to whether the dentist has an interest that extends beyond the nutritional requirements for the general health and development of the baby. He would seem to be justified, on occasion, in calling to the attention of mothers the advantages of nursing their babies for its effect upon both the health of the child and the more perfect development of the teeth. This is especially suggested in those cases in which a pediatrician is not directing the feeding of the baby.

FROM THE NURSING PERIOD UNTIL THE FIFTH YEAR. This is the most critical period during the child's life so far as the development of the teeth is concerned, as it relates to cellular activity in the building of the enamel and to gross structural defects; also in the nutritional damage to all cellular structures of the body and their relation to conditions which increase susceptibility to dental caries. The opportunity for the improvement of all of these conditions as a result of better nutrition is very great, and particular attention should be paid to the fact that the nutrition often requires special attention for months or years after an illness, to really restore a child to normal health and function.

It is during the period from birth to about the fifth year that practically all gross structural defects of the teeth occur. These represent severe shocks to cellular activity, and the nutritional damage is usually greater than during any other period of life; the effects are projected far into the future.

Here again the question may be raised as to the extent to which the dentist may be justified in giving advice regarding the diet. At the present time, the large majority of children are being fed without special direction of a pediatrician or trained nutritionist and it would appear to be the duty of the dentist who has made a special study of child health, nutrition and hygiene to give advice, or, in the absence of such qualifications, to recommend that a pediatrician be consulted.

AFTER THE FIFTH YEAR. In view of the fact that gross structural defects of the teeth very rarely result from periods of malnutrition occurring after the fifth year, the dentist's interest in nutrition should be almost entirely centered on those conditions of tooth environment which may influence susceptibility and immunity to dental caries. Increasing evidence is being accumulated in recent years of the importance of proper nutrition in this connection, yet, in view of the wide difference of opinion which prevails, it is without the province of this publication to discuss this question in detail. However, certain observations will be made in the following paragraphs.

GENERAL OBSERVATIONS ON HEALTH, NUTRITION AND HYGIENE,
IN THEIR RELATION TO ORAL PATHOLOGY.

The question may very properly be raised as to what extent the dentist is justified in advising patients with respect to nutrition. Physicians, and particularly pediatricians, are presumably better prepared to assume this responsibility. Even so, are there dental requirements in this field which are not receiving sufficient attention, or are they fully covered by the nutritional requirements for the general health? The dentist may also very properly inquire whether a trained person is directing the feeding of a child, and in specific cases should recommend that a pediatrician be consulted, or, if the dentist is qualified, he should himself give advice.

The following general observations should be kept in mind in connection with a nutritional program.

1. It is not practical to set up a diet list alone, for the reason that the problems of health, nutrition and hygiene are reciprocal and one should not be considered without the others. Certainly the physical condition of the child and the hygiene of eating, exercise and rest have much to do with the nutritional results of food eaten. The atmosphere of meal time should be restful — free from excitement of any kind.

2. Foods which supply nutritional requirements are well known, and satisfactory selections may be made from these, which include the foods generally best liked by children.

3. Diet regimentation applied to a number of persons may take appetite away, by causing some of the group to eat foods which they do not particularly like.

4. A standard daily requirement of food for children is impractical because their needs vary from day to day.

5. Hunger and appetite are the best guides in the selection of foods, and all of the food available for each meal, including the dessert, should be placed before the child at the beginning of the meal, in order that it may select what its appetite and hunger dictate.

6. The dentist should not attempt to set up nutritional programs for persons who are suffering from either systemic or generalized dietary deficiency diseases.

7. The dentist's efforts should be limited to the control, if possible, of oral conditions, on the premise that he may be able to influence tooth environment, but that tooth structure can not be changed after the teeth have erupted.

8. The dentist should understand how to obtain a satisfactory dietary case history and must be willing to take the time and trouble to obtain such information before offering advice or discussing changes in diet.

9. The dentist should be critical of his own activities and should avoid becoming a dietary faddist.

10. For practical purposes in considering a given case he should keep in mind the fact that the usual deficiencies encountered are protein, mineral and fat soluble vitamins. For the prompt correction of these (time is an important factor when these occur during the period of tooth development) he may and probably should prescribe:

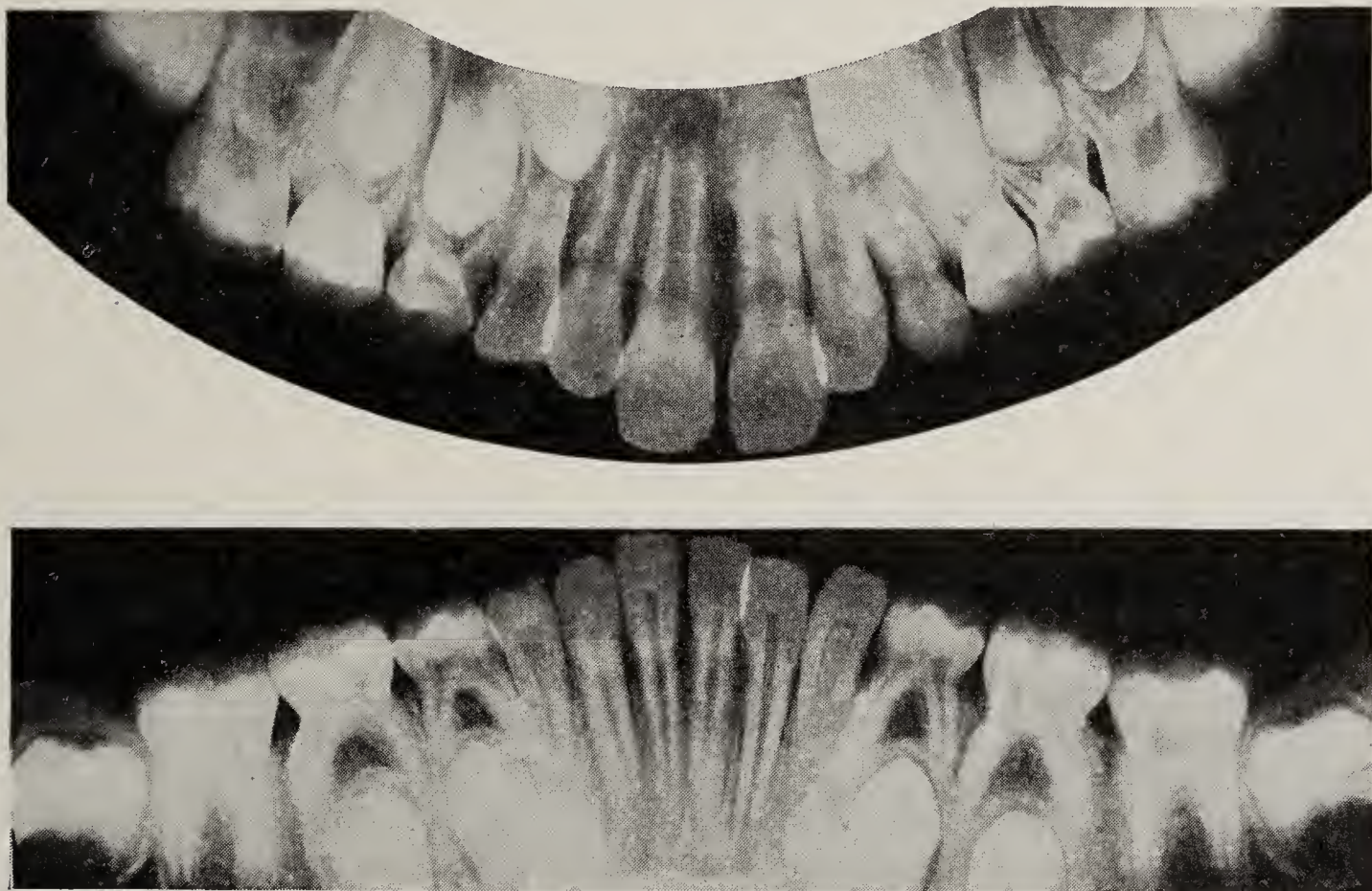


FIG. 7. Reproduction of radiographs of teeth of boy nine years of age who participated in the self-selection feeding experiment by Dr. Clara Davis from the time he was six months old and has continued on the same plan since the experiment closed. His teeth are entirely caries free.

These have been termed "panoramic" radiographic reproductions. Photographic enlargements were made of each film; these were cut to eliminate duplication of teeth and then pasted on a cardboard and rephotographed. They were retouched along the lines where they were matched together.

A high protein diet, in which liver, kidneys and eggs are predominantly featured.

Extra calcium and phosphorus in the form of dicalcium phosphate, *up to* 8 grams per day.

Cod liver oil, 12 to 24 cc. per day, using only products recognized by the Council on Therapeutics of the American Dental Association, or an acceptable substitute.

11. The dentist should avoid becoming overenthusiastic with respect to single additions to the diet, as a single mineral or a single vitamin. Calcium gluconate, for example, supplies only calcium and no phosphorus. Orange juice may be given in large amounts to remedy a supposed deficiency of vitamin C, but in doing this the consumption of other more important food constituents may be markedly decreased. The actual amount of the vita-

mins required to prevent deficiencies is not accurately known, but in general it is relatively small. In addition, less is known about the harm that may be done by overdosage of vitamins.

12. The dentist should realize that the building of sound bodies and teeth rest on the broad base of abundant biologically high grade foods, coupled with good hygiene, rather than on the addition to a *poorly chosen diet* of the particular adjuvants (vitamins or minerals) that cure or prevent a definite disease, such as rickets.

THE SELF-SELECTION FEEDING EXPERIMENT AND THE TEETH. Although the self-selection feeding experiment was undertaken by Dr. Davis without special consideration for its relation to or possible effect on susceptibility to dental caries, the fact should be recorded that, near the completion of the experiment, the teeth of the children were examined on two occasions by Dr. Corvin F. Stein, who reported that the teeth of all of the children were entirely free from caries. It is interesting to note, in this connection, that free sugar was excluded from the diet of these children and they were permitted to eat nothing between meals; also that sea-salt was substituted for the ordinary table salt.

Two of these boys, whose ages at this writing are 8½ and 9 years, respectively, have continued, as adopted members of Dr. Davis' family, to select their own food since the experiment closed, and their immunity to caries has been maintained. The radiographs of the teeth of the boy who is now nine are reproduced in Figure 7. Reference is made by Dr. Davis to the food eaten by these two boys during four consecutive "sample" days.

THE SALIVA

EARL A. ZAUS, M.D., AND HAROLD L. HANSEN, PH.D.

When the saliva is mentioned without designation of its special parts, it is most generally understood that the mixed fluid in the mouth is meant. This is made up of secretions from several sources, any of which may be examined separately. The three pairs of major glands include the parotid, sublingual and submaxillary glands. The accessory groups consist of very numerous, rather minute gland follicles, particularly in the mucous membrane of the lips, cheeks, tongue and palate.

SECRECTIONS OF VARIOUS GLANDS. Examination of the oral cavity reveals that it is constantly moist. This state is maintained by the accessory mucous and serous glands, which secrete continuously. The major glands function only intermittently and when stimulated. If the mucous membrane of the roof of the mouth is dried and the mouth held open for some time, one will generally find here and there globules of fluid collecting on its surface. Sometimes many of these will be seen; sometimes very few, and occasionally none without a very long wait. These tiny globules of secreted material may differ widely. Some readily show an acid reaction, and secretion may be so thick and viscous, that, by touching a finger to a globule, it may be drawn out into a very long thread. The larger accessory glands, such as those in the lips, emit a more serous type of fluid, presumably to keep the lips moist.

PHYSICAL CHARACTERISTICS. Not only are there variations in the viscosity of the individual droplets from these accessory glands, but also of the saliva as a whole. This is an observation that can be made by any dentist, and has been noted in practically all dental texts. Indeed, the general saliva may be almost as thick and viscous as mucus itself. In some instances, threads two or three feet long can be drawn out by touching a finger to the saliva and drawing it out with the finger. In filtering, this type of saliva leaves an unusually large amount of gummy material in the filter. Any stimulation of activity in the major gland group generally causes a thinning of the mixed fluid for a time.

Salivas not only differ with respect to viscosity, but also in their ability to hold their constituents in solution or suspension. Many people may be found whose teeth are generally free from deposits of gummy or slimy material. The saliva seems to hold not only all of its ingredients, but is also capable of dissolving quickly all of the ordinarily soluble things with which it comes in contact. Therefore such a mouth is always clean and free from

debris. In others, the saliva seems habitually incapable of holding its own ingredients in solution. The teeth especially are continually covered with a slimy viscid coating, and more solid deposits are frequently found.

Saliva of the kind just described has been called "ropy or viscous saliva." Generally the teeth will be very difficult to keep clean when the saliva is ropy. The person may, by care, keep the teeth looking well, but a close examination will show them to be covered by a transparent slimy material of sufficient thickness to be clearly apparent. If this be cleaned away, a new deposit will be found the next day. In many cases, if the saliva be touched by a rapidly rotating disk or stone, it will quickly thicken up into a more or less firm coagulum around the disk or stone. The degree to which this takes place is a rough measure of the ropiness of the saliva. If the attempt is made to prepare a cavity, it will be necessary to flush the mouth frequently with a considerable quantity of water to sufficiently remove the heavy saliva in order to be able to see the details of the cavity. While the degree of ropiness of the saliva is not a certain index to the degree of susceptibility to dental caries in the person, it will almost uniformly be found that caries occurring in the presence of this condition will be more difficult of management than when found in similar degrees in persons with thin, watery saliva. Proximal cavities, particularly, will more certainly spread broadly bucco-lingually and deposits on proximal restorations will be more likely to overstep the margins and cause recurrence of decay.

Another condition not frequently noted by writers, which may be frequently observed in clinical examinations, is the tendency to slimy or glutinous coatings upon the teeth of some persons, which, apparently, is a deposit from the saliva. These deposits may be found especially upon the axial surfaces of the teeth. They also tend to keep the mucous membranes slimy and slippery to the touch. There is also a degree of viscosity clearly discoverable by rubbing the buccal surfaces of the teeth back and forth a few times with the finger and then rubbing this material between the finger and thumb. This seems, often, to be independent of any considerable ropiness of the saliva. It seems to be a deposit from the saliva of a material which it fails to hold in solution. A related and possibly important discovery was made by a committee of the New York State Dental Society, which may be thus stated. The deposit is insoluble in the saliva of the person in whose mouth it is found, but is readily soluble in the saliva of persons in whose mouths such deposit is not found. It seems possible that this may favor the beginning of caries or actually furnish one of the necessary elements, by affording coverings that will shield microorganisms and their acid products from washings by the saliva; but, as yet, this is by no means assured. This condition

seems to be much less constant in the mouth of an individual than ropy saliva.

Every dentist should make careful observations of the qualities of saliva, which should be recorded and correlated with the conditions of the mouth and teeth. The value of this is not confined to diagnosis and prognosis in individual cases, but may also supply valuable information with respect to the etiology and prevention of dental caries and other diseases of the teeth and gingivae, especially if such findings can be harmonized with the results of adequate laboratory analyses.

CHEMICAL, PHYSICAL AND PHYSIOLOGICAL EXAMINATIONS

The preceding statements constitute a brief review of the general properties of saliva, as presented in the earlier editions of this work. There is little difference of opinion regarding them. By contrast, the status of knowledge concerning saliva based upon the best known methods of chemical, physical and physiological examination is in a highly controversial state. There are probably two reasons for this. Saliva is not a fluid like blood or urine with a relatively stable composition, but varies constantly over a wide range, even in the complete absence of any diseased condition. The methods of examination which have been found reliable and dependable for blood and similar body fluids do not always supply the same type of information for saliva. However, the investigations will, little by little, be brought into harmony with each other and with clinical conditions.

RATE OF ACTIVITY. Little is known about the rate of activity of the accessory glands, the composition of their secretion, or the nature or source of their stimulation. It is believed that they act continuously, but there is a wide variation between different individuals, and some degree of variation in the same person. The buccal, labial and anterior lingual gland groups are said to produce a mixed type of secretion. The posterior lingual group secrete a serous fluid and the palatine group secrete mucous.

THE TYPES OF STIMULI which determine the activity of the major glands are mechanical, thermal and chemical. These, of course, are directed toward the end of preparing the food for swallowing. Occasionally, when food is swallowed hurriedly and its descent is delayed in the oesophagus, a salivary reflex by way of the vagus nerve from the oesophagus to the salivary nucleus in the brain stem, is initiated. This has been referred to as the oesophago-salivary reflex. It is this reflex which is also responsible for the increased flow of saliva in the condition known as "heart burn." Other distant reflexes of this nature are to be found lower in the digestive system, i.e., stomach, small intestine, pancreas, liver, etc. This is to be remembered when one encounters a profuse salivary secretion in diseased states of these organs.

Psychic stimuli, such as the thought of food, influence the flow of saliva. Stimuli which excite the oral secretions may arise in various parts of the brain, particularly from the structure derived from the diencephalon.² An example of this is to be seen in the sialorrhea, that often appears early in the disease paralysis agitans, in which the pathology is to be found in the corpus striatum of the brain.

THE FUNCTIONS OF SALIVA are thought to be almost wholly mechanical. It aids mastication and swallowing, dilutes irritants, moderates the temperature of foods, and aids speech. It acts only indirectly in allaying thirst. The action of ptyalin plays a minor part in digestion and there are examples on record³ where its total absence produced no appreciable effect upon the digestive powers or nutrition of the individual. Finally, there is the controversial subject of the part saliva plays in maintaining the health of the hard and soft tissues of the mouth. Undoubtedly the buffering power of the saliva performs a protective function against decalcification of teeth by acids, such as those which are often found in citrus fruits.

SALIVARY COMPONENTS. The saliva is a slightly turbid, viscous fluid having a specific gravity of about 1.007 and containing in the neighborhood of 0.4 to 0.7 per cent solid matter. Of this solid matter a large proportion is organic in nature, the chief constituent being salivary mucin, with small quantities of albumin, globulin and enzymes.

Complete analytical data on the salivary components is meager. The available figures as well as reports made on individual components by various authors are not in complete agreement. This discrepancy is not only due to differences in analytical methods employed but also to variations in the composition of the salivary samples used.

H. Becks⁴ reported the following figures on the inorganic content of human resting saliva obtained from individuals having sound teeth and normal gum tissue and who were also in apparent good health:

Component	Mg. per 100 cc.	Method of Determination
Bicarbonate group	93.16	Van Slyke
pH	7.15	Quinhydrone
Potassium	55.89	Kramer-Tisdall
Calcium	12.13	Kramer-Tisdall
Magnesium	1.29	Kramer-Tisdall
Chlorin	74.68	Tschopp or Bang
Phosphate group	13.16	Tschopp or Belland Daisy
Sulphocyanate group	30.9	Colorimetric

Calcium and Phosphorous. Considerable work has been done in recent years on the calcium and phosphorous content of human saliva, but the results obtained have not been in agreement. Young-

berg⁵ reports 19.3 Mg. per 100 cc. for total phosphorous and 18.5 Mg. per 100 cc. for inorganic phosphorous. Entin and Geiken⁶ report 14 and 12, respectively, while Karshan and co-workers⁷ find in the neighborhood of 11 Mg. per 100 cc. for inorganic phosphorous.

The inorganic calcium content of the saliva apparently varies within fairly wide limits as is indicated by the work of Becks.⁸ (Personal communication from author). The variation is, however, not so great as in the case of phosphorous content. The results obtained by Becks may be correlated with the age groups of the subjects in the following way:

Age Group	Calcium Mg. per 100 cc.	Inorganic Phosphorous
5-20	2.7 to 7.5	6.7 to 30.4
20-40	2.3 to 10.95	6.3 to 32.
40-60	3.2 to 10.55	8.4 to 51.7
Over 60	2.2 to 10.2	6.1 to 36.6

It may be remarked that the extreme values for phosphorous are rather unusual and that nearly all analyses for that element give values in the neighborhood of 10 to 20 Mg. per 100 for inorganic phosphorous.

Updegraff and Lewis⁹ on the basis of rather extensive work on paraffin stimulated saliva obtained the data contained in the following table. The values obtained for saliva were compared with those of the blood from the same subjects. The concentration of nitrogenous substance in the saliva is on the average considerably lower than that of blood.

	Range Mg./100 cc.	Average
Total Solids	386-860	581
Non-protein Nitrogen	5.6-26.7	13
Ammonia Nitrogen	2.1-13.2	5.7
Ammonia + Urea Nitrogen.....	6.8-15.9	11.3
Uric Acid	0.5- 2.9	1.6
Urea Nitrogen	0.0- 6.7	4.1

The same authors point out that the salivary glands appear to be selective in that the blood components, glucose and aminoacids, are not secreted in significant amounts in the saliva, while urea and uric acid readily pass into the saliva. The ammonia in the saliva originates largely from the decomposition of urea. That the saliva is not a simple transudate or dialysate is also shown by the work of Beer & Wilson¹⁰ in their study of osmotic pressures and relative concentration of ions for blood and saliva. Some of the inorganic ions (K^+ Ca^{++} , HCO_3) are at higher concentration in parotid saliva than in blood and some (Na^+ Cl) are at lower concentrations.

Mucin. Of the organic components of saliva the mucin has not received so much attention as the subject undoubtedly deserves. Inoye¹¹ reports that salivary sodium mucinate is completely

soluble in water, occurs to the extent of about 0.25 Mg. per 100 cc. in resting saliva and is not precipitated by salts of lead and mercury in the biological range of concentration. The iso-electric zone for salivary mucin is the pH range from 2.95 to 2.75, a greater hydrogen ion concentration than for gastric mucin, pH 4.98. According to Miller and Dunbar,¹² the viscosity of gastric mucin is greatest at its iso-electric point. Both salivary mucin and gastric mucin appear to belong to the same class of compounds, the conjugated proteins, and both of them give the tests for carbohydrate and for protein.

Calcium and Phosphorous Ions. A study of the composition of saliva, as well as other experiments, shows that normally the saliva is at least saturated with calcium and phosphate ions. If it were not for this fact the teeth would slowly dissolve. In addition, the saliva exerts buffering power against both alkalies and acids. On the average, unstimulated saliva is equivalent to .017 normal NaOH while stimulated saliva is equivalent to about 0.025 normal NaOH.¹³

Composition Inconstant. It has already been noted that the composition of saliva is inconstant. This is particularly true of stimulated saliva. However, the trend of the available evidence seems to indicate that it is not practically possible to change the composition of saliva for any definite length of time by artificial means. Clark and Levine¹⁴ report no constancy in salivary calcium, phosphorous, chloride and ash under regulated dietary conditions nor during fasting. They noted that abnormal ingestion of inorganic phosphates produced a marked excretion of phosphate in the saliva. Clark, Shell, Josephson & Stockle¹⁵ found no relationship between quantity of given element ingested or retained and the quantity in the circulating blood or resting saliva or between the concentration of inorganic constituents in the blood and saliva.

Correlation with Dental Caries. From time to time various writers have made attempts to correlate the composition of saliva with the incidence of dental caries and also to correlate changes in salivary composition, particularly calcium content with diet. The results obtained are so conflicting and in many cases the experimental evidence is so obscure that one is forced to the conclusion that it is impossible at the present time to make definite statements on the subject. The weight of experimental evidence, however, is against the existence of any such relationships. This statement is substantiated by the work of Hubbell.¹⁶

Determination of pH. Recent developments in methods of pH determination have made it apparent that many of the data obtained on the pH of saliva are in error, particularly those based upon the use of test papers. The quinhydrone method is also un-

reliable. So far, no extended reports on the pH of saliva by means of the glass electrode method are available.

While subject to error, the use of the indicator method in the hands of competent experimenters shows that saliva is more often acid than alkaline. Starr¹⁷ found the pH of resting saliva varied from pH 5.75 to 7.05; in 86 per cent of the cases it was between 6.35 and 6.80. The mean, mode and median coincided at pH 6.60. His work indicates that the pH of saliva is not directly related to that of blood. The results by Hermann¹⁸ are in substantial agreement with the values given by Starr.

From time to time, reports have appeared in the literature purporting to show variations in composition and in pH of the saliva due to various pathological conditions. At the outset it should be remarked that any data of this type should be given most careful observation. Inasmuch as the composition of saliva as well as its pH normally vary within wide limits and also because of the fact that there are differences between resting and stimulated saliva, it is extremely difficult to determine when and if the variations noted are abnormal or pathological in origin.

Sullivan and Jones¹⁹ report that in Pellagra the saliva is ropy, of a high mucin content, has a high specific gravity and tends to be more alkaline than normal. Some authors claim that sugar is found in the saliva of diabetics; this has been denied by others. The same sort of statement may be made concerning the presence of excessive amount of urea in the saliva of patients with renal disorders. Entin and Geiken⁶ claim that variations in salivary inorganic phosphorous run parallel with the phosphate in the blood serum. Phosphate content is increased in tuberculosis of the bone, diabetes and nephritis and is decreased in syphilis.

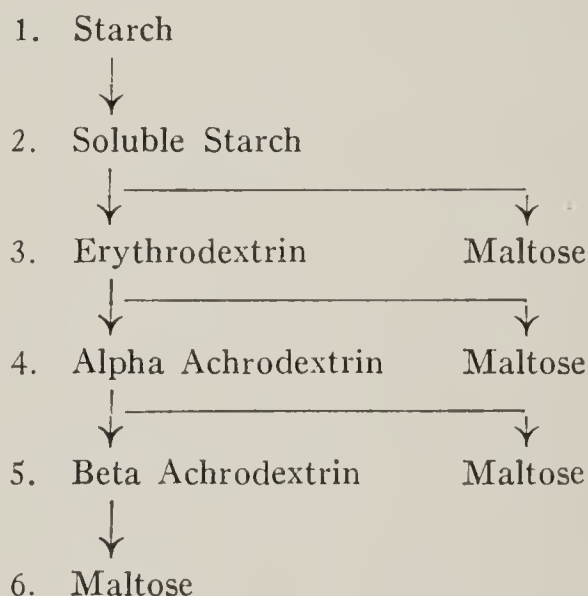
Enzymes. The enzymes which have been reported as present in the saliva are ptyalin, catalase, maltase, lipase, urease and a protease. All but the first are found only in traces and it should be remembered that the source or importance of these enzymes occurring in traces is not definite. The presence of bacteria, food residues, desquamated epithelium and leukocytes must be considered as a possible source. A phosphatase has also been reported as occurring in small amounts in saliva.²⁰ It is probably derived from gum tissue.

The salivary amylase in man occurs even at birth, but increases in concentration during childhood. In adults the amount of salivary amylase in the saliva is extremely variable. Mayer²¹ has published an interesting report on this subject.

Ptyalin acts in fluids over a wide range of hydrogen ion concentration, pH 4 to 9. It is most effective at a pH of 6.5. This is contrary to the somewhat common notion that it acts only in an alkaline medium. It, therefore, continues its action in the stomach until the pH of the substrate becomes 4. This frequently takes an

appreciable length of time.²² In vitro, saliva rapidly loses its amylolytic power, although preservation with glycerol tends to maintain some activity for a year or more,²³ but once acidified its activity cannot be restored by neutralization.

Ptyalin acts readily on cooked starch and slightly on raw starch. Glycogen is hydrolyzed by the salivary amylase of omnivora including man, but not by that of herbivora. In the course of starch hydrolysis, a series of polysacharides of steadily decreasing size is formed. There is some evidence to indicate that maltose is split off in each stage of the action of saliva since it appears very early in the digestion. Although the exact course of the digestion is unknown the following representation is probably somewhat near the truth:



The salivary amylase requires some negative ion as an activator. The following are effective as coenzymes in descending order: Cl, Br, NO₃, SO₄, PO₄, CH₃COO.

The relationship of amylase content of the saliva to dental caries is uncertain. Some authors claim to have found such relationships but are in disagreement as to whether the amylase content is high with a high caries incidence or vice versa. Prinz²⁴ reports there is no relation between dental caries and amylase content.

Delourge²⁵ reports that gastric hypoacidity is associated with a low ptyalin content and hyperacidity with a high ptyalin concentration. Carles and Delmas-Marsalet²⁶ find that in infectious diseases as well as in various conditions of cachexia the amylolytic power of saliva is diminished. It is also very clearly affected in pathological conditions of the stomach. According to Gayda,²⁷ insulin causes a great increase in the amylase activity of saliva, being increased as much as seventeen times after insulin injection. This effect wears off with time.

Among the drugs which have been found to be excreted in the saliva are the following: potassium iodide, arsenicals, cocaine, morphine, heroin, quinine and strychnine.²⁸

DISTURBANCES IN THE RATE OF SALIVARY SECRETION. The minute rate of flow of saliva is subject to extreme variations. Data accumulated in the physiological laboratories of Northwestern University Dental School show the resting rate of flow in normal subjects to be about 0.5 cc. per minute, while the rate of flow following stimulation by chewing gum or paraffin was found to vary from 0.9 cc. to 9 cc. per minute, the greatest number of individuals falling around 2.5 cc. per minute. The daily output of saliva is usually from 1 to 1½ liters, but under certain pathological conditions, as much as 10 liters per day has been reported.

When the rate of flow of saliva becomes considerably diminished, difficulty in speech and swallowing occur. Various names have been applied to this clinical condition of dry mouth, such as xerostomia, aptyalism and oligo-sialia. Dry mouth may result from all causes of body dehydration, such as excessive perspiration, fever, vomiting, diarrhea, hemorrhage, and polyuria. Fright is capable of producing a reduction in salivary flow, as many public speakers can attest. A nervous type of xerostomia (Zagaris' disease) has been described. It occurs usually in nervous women at the menopause, often follows a severe shock, is apt to be more or less permanent in character, and is thought to be due to a lesion in the salivary nucleus located in the brain stem.

A congenital form of "dry mouth," due either to dysfunction or absence of the salivary glands has been reported.³ Often this may be associated with a similar disturbance in the lacrymal glands. These cases come to the attention of the dentist because there is usually rampant destruction of the teeth, seen first in the temporary and later in the permanent teeth.

An increased rate of flow of saliva (sialorrhea, ptyalism, salivation, sialosis) occurs in many conditions. The daily output may amount to 3 to 4 liters, and sometimes as much as 10 liters. The causes of salivation may be listed as follows:²⁹

1. Drugs or poisons; mercury, bismuth, iodides, bromides, arsenic and copper.
2. Local irritation in oral cavity; infections, scurvy, sprue, purpura anemia; teething, epulis, salivary calculus and poor fitting dental appliances.
3. Infectious diseases, especially small pox and rabies.
4. Reflex stimulation from oesophagus, stomach, pancreas, liver and uterus. It is particularly marked in carcinoma of the cardiac end of the oesophagus.
5. Diseases of the nervous system, such as sea sickness, car sickness, hysteria, migraine, and the more serious organic diseases such as irritation of the chorda tympani usually with paralysis of the seventh cranial nerve, degeneration lesions of the basal ganglia as in paralysis agitans and encephalitis, lethargica, tic douloureux, and tabes with salivary crises.

6. Idiopathic cases in which no cause can be found, developing after severe shock or emotion.

SALIVA AND FETID BREATH. There is but little evidence to indicate that the saliva is directly responsible for fetid breath. Haggard and Greenberg³⁰ have shown that the odors present after eating alliaceous substances (garlic, onion, etc.) are not due to the excretion of these substances in the saliva but are due solely to particles retained in the structures about the mouth, particularly in the grooves and papillae of the tongue. In certain diseased states, such as nephritis with uremia, urea is excreted through the saliva and is broken down to ammonia in the mouth, giving rise to the characteristic ammonia breath of these patients. It is likely also that saliva may be attacked by bacteria in diseased mouths and so give rise to offensive odors. As a rule, however, fetid breath is due to conditions local to structures of the mouth other than saliva, or to conditions in the respiratory tract.

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Development of the Teeth and Their Supporting Structures

20 ILLUSTRATIONS: FIGURES 8A-9s.

Soon after the fourth week of the embryo, the development of the mouth and face begins. The fore-gut forms the pharyngeal portion of the mouth. Paired lateral outgrowths from the fore-gut form the right and left sides of the mandible, which join at the symphysis; they also form the lateral portions of the maxillae, each of which eventually contains the teeth forward to and including the cuspid. The incisal portion of the maxillae is a downgrowth from the forebrain. See Figures 8A and 8B. These outgrowths and the downgrowth, which form the jaws, are mostly mesoderm—

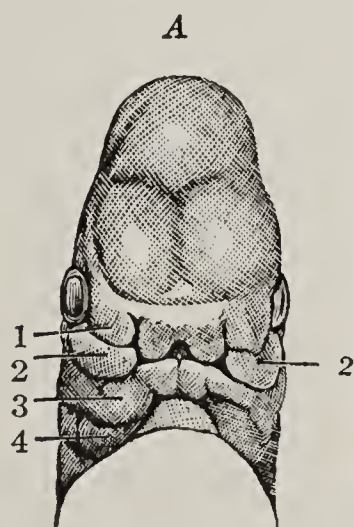


FIG. 8A.

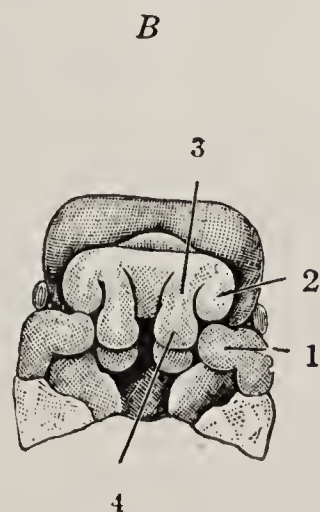


FIG. 8B.

FIGS. 8A and 8B. Embryo. A, front view, frontonasal process and maxillary process about to unite to form the maxillae: 1, lateral nasal part of frontonasal process; 2, maxillary lateral outgrowth; 3, mandibular lateral outgrowth; 4, the hyoid arch. B, same embryo with the mandibular arch removed. 1, maxillary lateral outgrowth; 2, lateral nasal process from downgrowth from forebrain; 3, mesial nasal process; 4, downgrowth from forebrain to form the incisal portion of the maxillae. (Noyes Histology.)

connective tissue. An infolding of the ectoderm forms the forward portion of the mouth and provides an epithelial lining, from which the teeth are developed. The teeth are, therefore, appendages of the skin, as are the hair and nails. This is, at first, a combined mouth and nose cavity, and an opening develops between it and the pharyngeal portion, thus connecting the mouth with the alimentary

tract. The mouth and nose are later separated by the development of the hard and soft palates.

TOOTH DEVELOPMENT begins about the sixth week, when there is a multiplication and inward growth of a cord of epithelial cells from the crests of the jaws. This is the dental lamina, from which there occur inverted cup shaped proliferations to form the enamel of the crowns of each of the temporary teeth, and at a later time

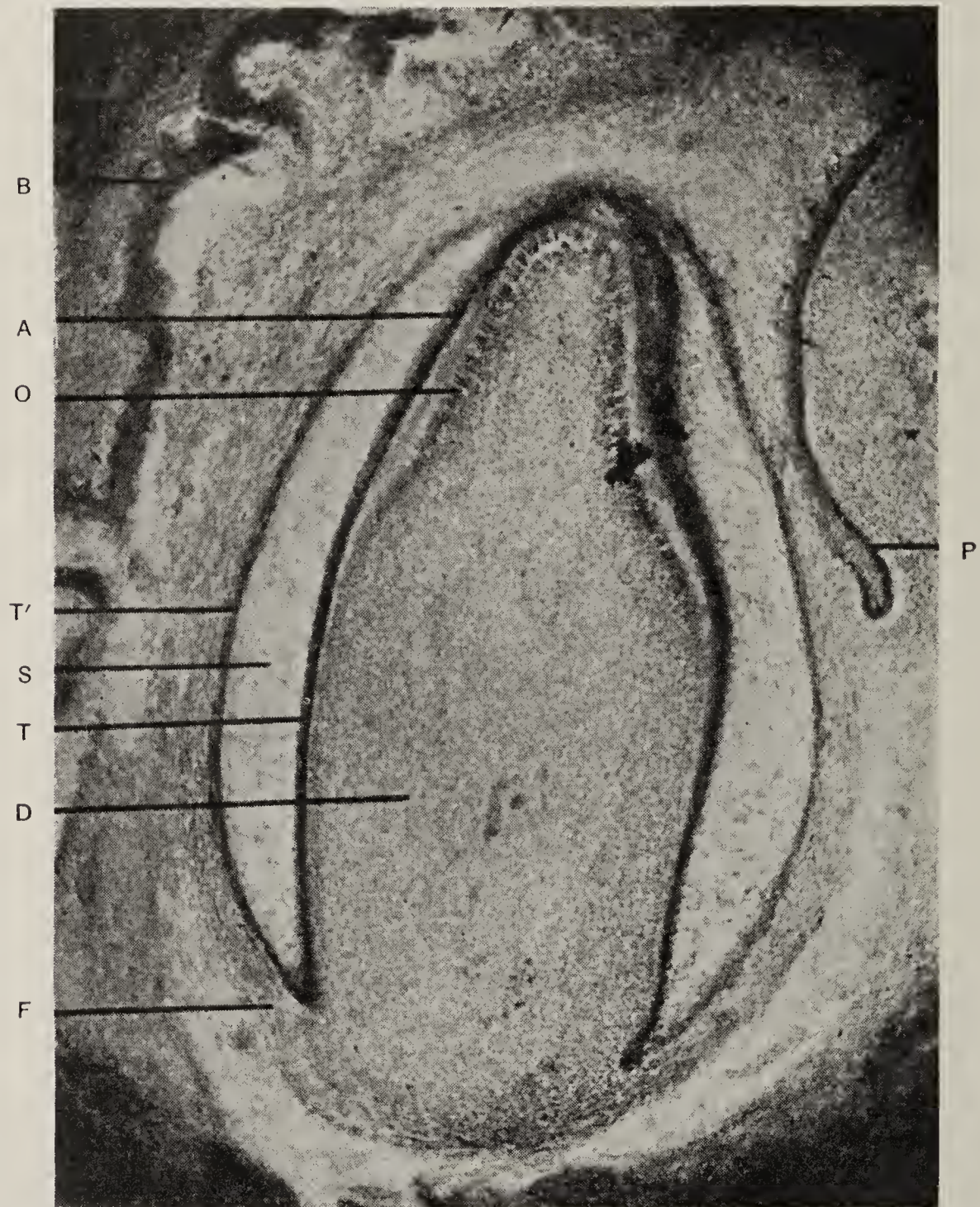


FIG. 8C. The tooth germ, showing the bud for the permanent tooth at P. Calcification is just beginning. F, follicle wall; D, dental papilla, T, inner tunic; T', outer tunic; S, stellate reticulum; O, odontoblasts; A, ameloblasts, B, bone. (Noyes Histology.)

additional proliferations occur for each of the permanent teeth. See Figure 8C. Infrequently, abnormal proliferations occur, which result in the formation of supernumerary teeth. The cup shaped epithelial structure, which becomes separated from the cord, is the enamel forming organ. A connective tissue growth into each cup—the dental papilla—forms the dentin and then persists as the pulp of the tooth. The tissue around the dental papilla becomes

the dental follicle, which forms the cementum and peridental membrane.

The process and progress of tooth development is of importance in relation to problems which arise in connection with the pathology of dental caries and dystrophies which involve the enamel and dentin, also in connection with the diseases of the dental pulp and the pericemental structures.



FIG. 9A.

FIG. 9B.

FIG. 9A. Diagrammatic representation of occlusal view of dentin islands of lower first permanent molar at birth.

FIG. 9B. Diagrammatic representation of growth of dentin islands at one year of age.



FIG. 9C.

FIG. 9D.

FIG. 9C. Diagrammatic representation of growth of dentin islands at two years of age.

FIG. 9D. Diagrammatic representation of joining of dentin islands at about the third year to complete the outer surface of the dentin crown.

DENTIN CALCIFICATION. The first actual calcification of tooth structure is dentin, which is built by the outer layer of the cells of the dental papilla—the odontoblasts. A small island of dentin is formed in the position of each “dentin cusp,” which is the foundation for the formation of each enamel cusp or lobe. For the lower permanent first molar, for example, five dentin islands are formed, each directly under the position of a future enamel cusp. These five areas of dentin gradually become thicker and

enlarge laterally until they join together and eventually form the dentin crown of the tooth. Figures 9A, 9B, 9C and 9D illustrate diagrammatically about the average formation and growth of the dentin islands of this tooth at birth, and at one, two and three years.

ENAMEL CALCIFICATION. Soon after the islands of dentin are formed, the cells which line the enamel organ—the ameloblasts, deposit a little mound of enamel on each dentin island. See Figure 9E. This is followed by layer after layer of enamel, as indi-



FIG. 9E. The dentin and enamel layers, showing a deposit of a thin layer of enamel, indicated in black, supported by a much thicker layer of dentin. The ameloblasts are shown above the enamel, the odontoblasts below the dentin. (Meyer's Histology and Histogenesis of the Teeth; Churchill.)

cated by the lines of Retzius, each in the form of a cap which fits over that previously deposited. The dentin is always formed in advance of the enamel, and the calcification of enamel continues until the five islands coalesce and finally the entire enamel crown is completed. The last layers of enamel are added on the sides only of the several cusps; these are not complete caps. See Figures 9F to 9S, which constitute a series of eleven diagrammatic mesio-distal sections of the lower first molar through the three buccal cusps, the crown and roots, also one photomicrograph of the formation of the gingivae as the tooth erupts (Figure 9M).

These illustrations show: (1) The growth of the dentin islands at birth and about the average growth of dentin for each year up to ten, when the root is generally completed; (2) the

amount of enamel deposited at birth and the average annual additions which complete the crown about the fourth year; (3) the building of cementum, which is deposited in more or less definite layers and covers the root portion of the dentin; also the formation of the peridental membrane and alveolar process. (4) the formation of the enamel cuticle, the change in the ameloblasts, after the enamel is completed, and the subsequent union of the enamel epithelium with the mouth epithelium just before the tooth



FIG. 9F.

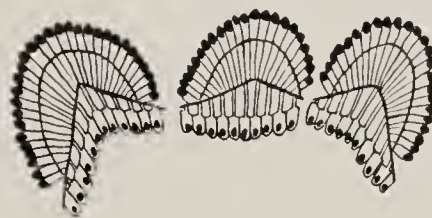


FIG. 9G.

FIGS. 9F to 9R. A series of drawings to represent the annual progress of calcification of the dentin, enamel and cementum of a lower first molar; also the development of the gingivae, peridental membrane and alveolar process. These are vertical mesio-distal sections, presumably through the three buccal cusps, then through about the center of the pulp chamber and roots.

FIG. 9F. The formation of dentin and enamel at birth. There are three dentin islands, one for each cusp, with a row of odontoblasts on the pulpal (lower) surface; also three mounds of enamel, with a row of ameloblasts on the outer (upper) surface.

FIG. 9G. The extent of the growth of the dentin and enamel at one year.



FIG. 9H.

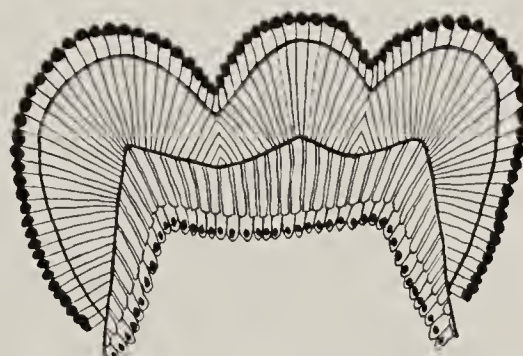


FIG. 9J.

FIG. 9H. The extent of the growth of the dentin and enamel at 2 years.

FIG. 9J. The growth of the dentin and enamel at 3 years.

erupts, thus forming the crest of the gingivae, which recedes and encircles the crown of the tooth as it erupts.

GROWTH OF DENTIN INWARD; THE ODONTOBLASTS. It will be noted that the dento-enamel junction, throughout the entire crown, is the place of the beginning formation of both the dentin and the enamel; the growth of the dentin is inward; the papilla becoming smaller and smaller as the dentin becomes thicker and thicker, until the dentin crown and pulp are of normal dimensions. The outer layer of cells of the pulp—the odontoblasts, persist through-

out life, or the period of pulp vitality. The dentin is built in the form of tubules, which extend from the dento-enamel junction to the pulp. There is a tubule which corresponds to each odontoblast and there is a projection of the odontoblast which occupies the center of the tubule and transmits pain sensation, although it is not a nerve fibre. The odontoblasts are often stimulated to renew their activity of dentin building under any one of several conditions by which they are irritated. In some cases they continue to build dentin until only a slight thread of pulp tissue remains, and this gradually dies—apparently a degenerative process. Normal dentin contains neither blood vessels, nerves, nor lymph vessels.

GROWTH OF ENAMEL OUTWARD; THE AMELOBLASTS. The enamel, on the other hand, is built from within outward. The enamel organ gradually enlarges as the building progresses, and when the enamel

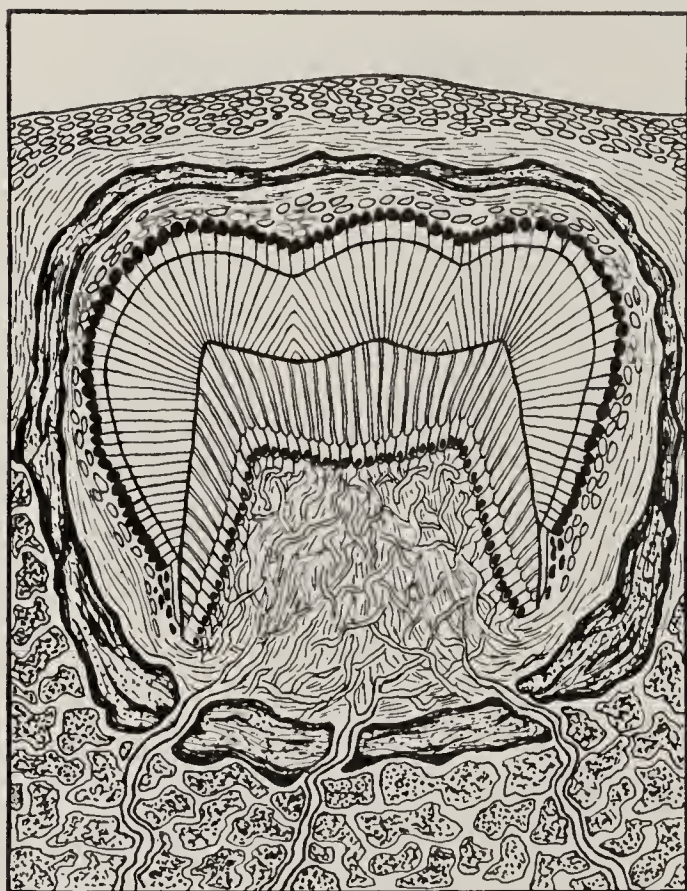


FIG. 9K.

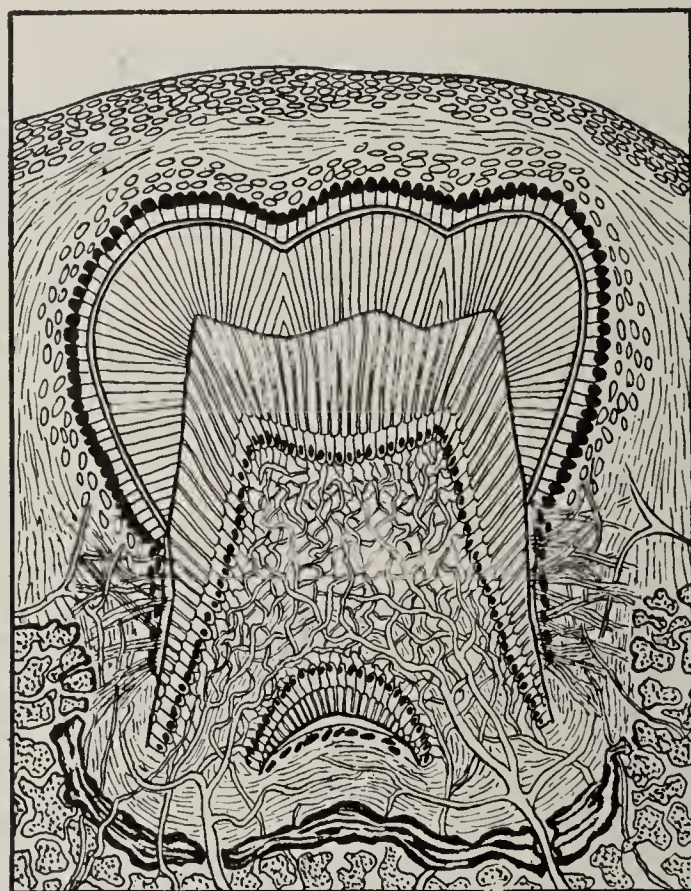


FIG. 9L.

FIG. 9K. At 4 years the enamel crown is represented as being completed (it should be understood that there may be considerable variation as to the time when this occurs.) The ameloblasts remain in their positions to form the enamel cuticle. The odontoblasts have formed a little of the dentin root, and a thin layer of cementum has been deposited on this by cementoblasts, which lie in the tissues on the outer surface of the root.

FIG. 9L. At 5 years, the enamel cuticle is completed and the ameloblasts then change to the more common type of epithelial cells which surround them. They are separated from the gum or mouth epithelium by a rather thin layer of connective tissue. More dentin has been formed, including an extension of the roots, also a beginning of the floor of the pulp chamber. Additional cementum has been deposited and a little of the bone of the alveolar process has been formed.

cap is completed, the enamel forming cells construct the enamel cuticle and change back to the more common type of epithelial cells from which they came. As ameloblasts, their function is fulfilled when the crown of the tooth is completed and they then disappear. There can be no further development of, or changes in the enamel, as an activity of the formative organ.

DEVELOPMENTAL LINES OF THE ENAMEL. The lines along which the several lobes join to form the crown of each tooth are known as "developmental lines." In some cases these lines are not apparent as one views the completed crown, as for example the labial surface of each incisor is made up of sections of three lobes of enamel, with developmental lines which divide it into mesial, middle, and distal thirds, yet the contour of the surface may be so smooth as to give no suggestion of the positions of the developmental lines. Their positions are often apparent where they cross the incisal edge. In many positions, and particularly on the occlusal surfaces of the bicuspid and molars, the developmental lines are smoothly depressed in the form of slight grooves, and a smoothly rounded fossa is formed where three or more grooves meet. These grooves and fossae are the normal forms of these teeth. The enamel contains neither blood nor lymph vessels nor nerves.

PITS AND FISSURES IN THE ENAMEL. In certain positions in several of the teeth, the lobes of the enamel oftentimes fail to unite perfectly and there are defects in the form of pits or fissures, in the positions of grooves and fossae. Pits occur on the occlusal sur-



FIG. 9M. A photomicrograph showing the beginning of the formation of the gingivae as the tooth erupts (in this case a temporary molar), the enamel epithelium joins with the mouth epithelium and the gum tissue gradually recedes about the sides of the crown to form the gingivae. (Meyer's Histology and Histogenesis of the Teeth; Churchill.)

face of the bicuspid and molars, also on the buccal or lingual surfaces of the molars, or on the lingual surface of the upper lateral incisors. Fissures may occur on the occlusal surfaces of the bicuspid and molars and occasionally these extend through the marginal ridges of molars to the buccal or lingual surface. Pits and fissures present opportunity for lodgements of food debris and microorganisms and thus promote the occurrence of decay.

THE DENTIN ROOT. When the outer surface of the dentin crown is completed, the odontoblasts continue the building of the dentin root; the outer surface of the dentin at any level is always formed first, and the central partially formed portion of the root is more or less funnel shaped, with the large end of the funnel in the position of the end of the forming root. The root becomes gradually smaller as the apex is approached and finally, as the dentin of the root apex is formed, the opening is reduced to a small foramen, through which blood vessels, lymph vessels and nerves reach or make exit from the pulp tissue. Occasionally there are two or more foramina at the root apex.

THE CEMENTUM is gradually laid down upon the dentin, and follows the dentin in its development toward the apex, eventually covering the external surface of the dentin root. Like the enamel, the cementum is built in layers, although more irregularly, from the inside outward, but unlike the enamel, the cementoblasts of

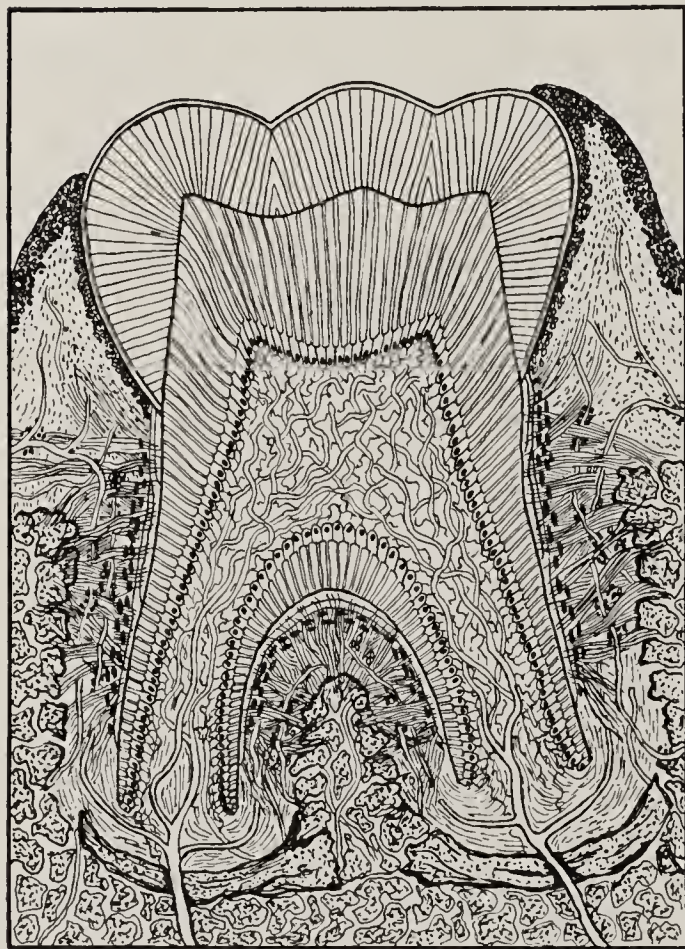


FIG. 9N.

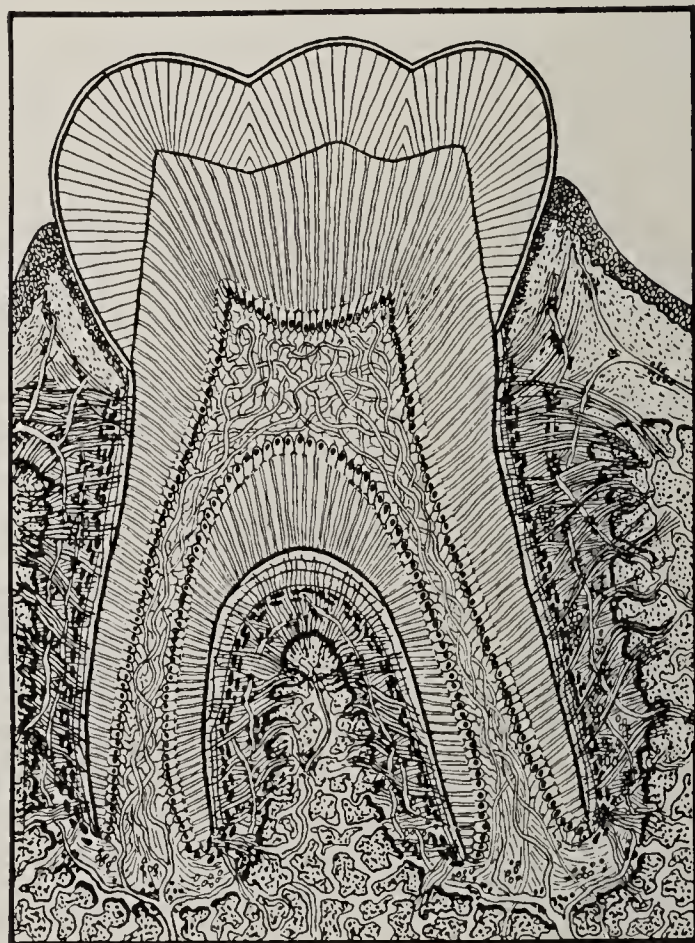


FIG. 9P.

FIG. 9N. At 6 years the tooth pushes through the gum. Before it breaks through, the ameloblasts all disappear and the enamel epithelium joins with the mouth epithelium over the occlusal surface of the crown. As the tooth erupts, the soft tissue gradually recedes to form the gingivae. Dentin building by the odontoblasts continues; the pulp chamber is reduced in size and the roots begin to take form; the cementum on the surface becomes thicker as the cementoblasts continue to build cementum. The formation of the periodontal membrane and the bone of the alveolar process follows the tooth as it erupts.

FIG. 9P. At 7 years the dentin of the crown portion is very nearly completed, the root is becoming longer, and the canals smaller. The cementum, periodontal membrane and alveolar process continue their growth.

the dental follicle which build it, do not disappear, as do the ameloblasts, and additional layers of cementum may gradually be added

throughout life. These cells lie within the peridental membrane, and may be stimulated to build excessively thick cementum. There are no blood vessels, lymph vessels or nerves in the cementum. It therefore has no sense of pain and can not participate in any inflammatory reaction.

THE GINGIVAE. It will be noted that formation of the gingivae begins with a union of the enamel epithelium with the mouth epithelium, previous to the eruption of the tooth; this is followed by the recession and condensation of these soft tissue flaps in all directions to become an encircling membrane about the crown as it erupts. See Figure 9M. Principal fibres of the peridental membrane are then projected into the gingivae to give them support. The gingivae are therefore composed of connective tissue, with an

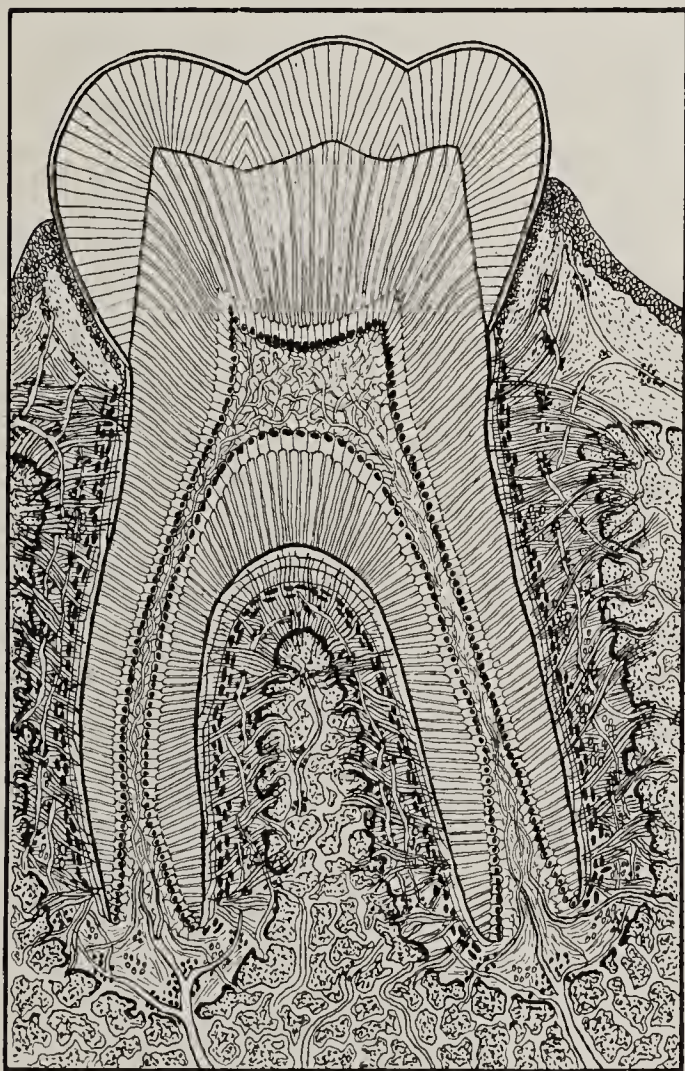


FIG. 9Q.

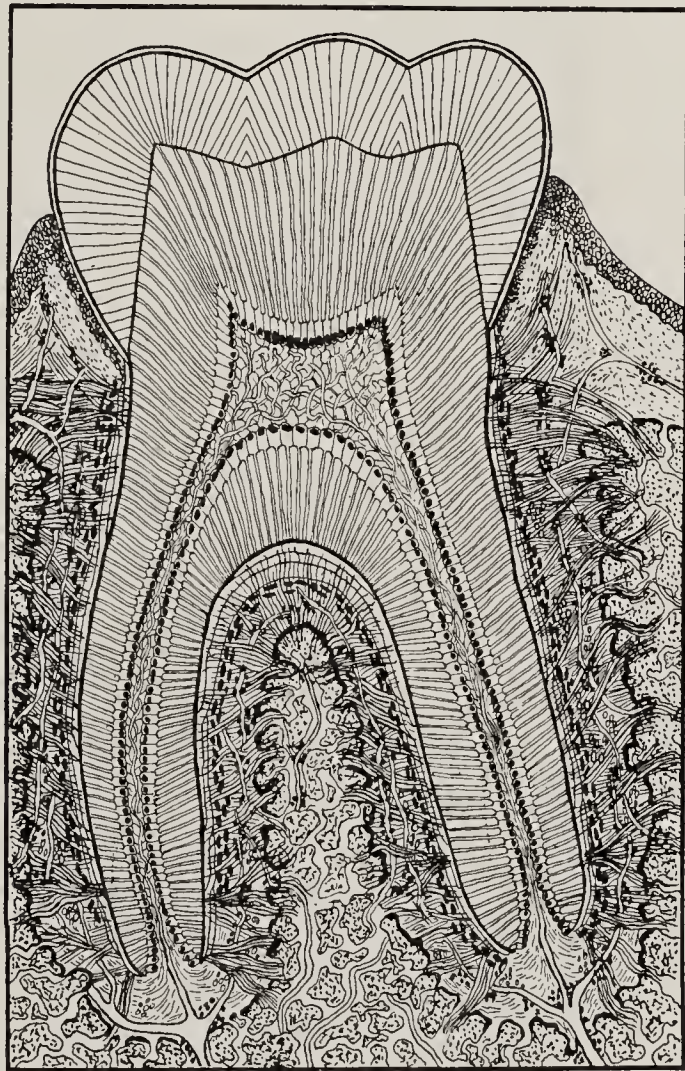


FIG. 9R.

FIG. 9Q. At 8 years the continued activity of the odontoblasts has further reduced the size of the pulp chamber and root canals and the root is nearing its full length. The building of cementum, peridental membrane and alveolar process continues.

FIG. 9R. At 9 years the roots have reached about their full length, but the apical foramina are still wide open, so that good root fillings could not be made. The pulp chamber and the root canals are approximately completed, except the apical portion.

inner covering of epithelium in contact with the enamel and an outer covering of epithelium which is continuous with that of the gum. The word gingiva is derived from the Latin *gigno*, meaning "to be born." The crown of the tooth erupts, or is born, through the gum, and the gum which encircles the crown is called the gingiva, or plural gingivae.

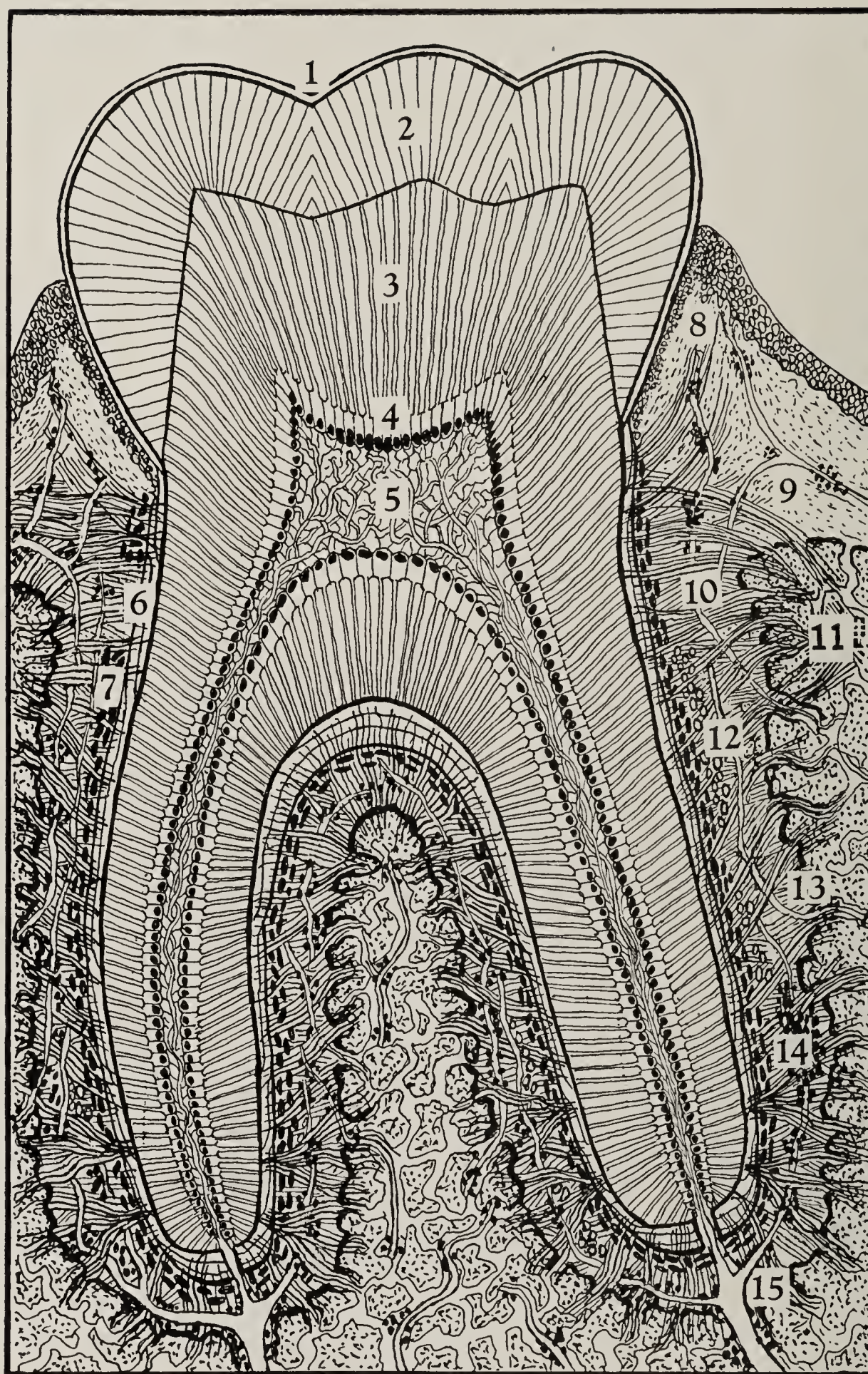


FIG. 9s. At 10 years the tooth is completed. The apical foramina are small, cementum covers the entire outer surface of the roots and is everywhere connected to the bone by fibres of the periodontal membrane. Blood vessels, nerves and lymphatics traverse the tissues of the pulp, periodontal membrane and bone to maintain the vitality of the odontoblasts and cementoblasts, also of the fibres and other structures of the gingivae and periodontal membrane and of the bone to which the fibres are attached. Some fibres extend into the gingivae and others reach across each interproximal space to hold the teeth in close contact. All of these structures will be more critically examined as the pathology of the various tissues are studied.

Explanation: 1, enamel cuticle; 2, enamel; 3, dentin; 4, odontoblasts; 5, pulp; 6, cementum; 7, cementoblasts; 8, gingivae; 9, blood vessel from gum to periodontal membrane; 10, principal fibres of periodontal membrane; 11, bone of alveolar process; 12, epithelial cells close to cementum; 13, blood vessel from bone to periodontal membrane; 14, lymphatic vessels attached to blood vessels; 15, blood vessel from apical region into periodontal membrane.

THE PERIDENTAL MEMBRANE. The principal functions of the peridental membrane are: (1) to maintain the attachment of the tooth root to the bone of the alveolar process; (2) to cushion the tooth against the stresses of mastication, as applied to the crown either laterally or in the direction of the long axis of the tooth; (3) to give support to the gingivae. The principal fibres of the peridental membrane are imbedded in the cementum and extend either occlusally into the connective tissue of the gingivae or in various directions to the bone of the alveolar process; some fibres in the gingival region connect the proximal surfaces of adjoining teeth. The variations in the directions of groups of fibres is such as to best withstand and cushion the tooth against the several stresses which are brought upon the tooth in mastication. The peridental membrane is almost entirely made up of connective tissue, although it contains clusters of epithelial cells. It is well supplied with blood vessels, lymphatic vessels and nerves. See Figure 9S.

THE ALVEOLAR PROCESS is an extension of the bone of the jaw to support the tooth, by maintaining the positions of the outer ends of the principal fibres of the peridental membrane which are attached to it. The alveolar process is projected farther and farther from the jaw bone proper as the root of the tooth develops and the tooth erupts. It disappears when the tooth is extracted, or over any given area when the fibres of the peridental membrane are detached from the cementum, because it can no longer perform its function of supporting the tooth.

The minute structure of the enamel, dentin, pulp, gingivae, peridental membrane, cementum and alveolar process will be separately discussed in connection with the consideration of the diseases and treatment of these tissues.

The Temporary and Permanent Dentitions

CALCIFICATION: ERUPTION: OCCLUSION.

17 ILLUSTRATIONS: FIGURES 10A-14B.

In preparing diagrams representing the average time of beginning calcification and the complete calcification of the enamel, the completion of the roots, and the time of eruption of the several teeth, also the average time of beginning absorption and shedding of the temporary teeth, it should be remembered that there are many variations from any schedules which may be set up. It seems better to give average figures, rather than both extremes, except for the time of completion of the roots of the permanent teeth. In all cases in which there is any doubt as to the exact condition, the determination should be made with radiographs.

There are considerable differences in the findings of investigators, particularly as to the time of beginning calcification and the completion of the crowns. In revising the data presented in the first edition of this work in 1908, the publications by Noyes¹, Churchill² and Kronfeld³ have been consulted, also a radiographic study by Bengston⁴. Records of a considerable number of cases of hypoplasia have also been studied to check on the contemporaneous calcification of the several teeth. The bicuspid and second molars so rarely show hypoplastic defects that the conclusion may be fairly drawn that the calcification of these teeth must begin, as a rule, after the time when a period of malnutrition will effect the upper incisors, cuspids and first molars.

THE TEMPORARY TEETH.

CALCIFICATION. The temporary teeth of one side of the upper jaw are represented in outline considerably enlarged in Figures 10A and 10B. In the first of these, the calcification of the crowns and the roots of the teeth is represented by figures placed upon the individual teeth. In the second, the absorption of the roots is represented in a similar way. The two, taken together, give a brief

¹ Noyes; *Dental Histology and Embryology*, fourth edition, 1929, Lea and Febiger, Philadelphia.

² Meyer's *Normal Histology and Histogenesis of the Human Teeth and Associated Parts*, translated and edited by Herman R. Churchill; 1935, J. P. Lippincott Co., Philadelphia.

³ *Histopathology of the Teeth and Surrounding Structures*, Rudolph Kronfeld; 1933, Lea and Febiger, Philadelphia.

⁴ *A Study of the Time of Eruption and Root Development of the Permanent Teeth*, Ralph G. Bengston; 1930, Thesis submitted in part fulfillment of requirement for Master's degree, Northwestern University Dental School.

synopsis of the changes which take place, together with the average time in years occupied. The calcification of the temporary teeth, according to this chart, Figure 10A, begins in the central and lateral incisors at about the seventeenth week of uterine life, in the first molar at about the twentieth week, and in the cuspid and second molar at about the twenty-second week. The position of 0 on the various teeth indicates the average calcification at birth, the figures 6, 12 and 24 represent, in months, the average progress of the calcification of each tooth. The figures above the root apices indicate, in years, the average time of completion of the roots. It must be understood that these figures are averages from which there are wide variations; also, that they have little to do with the time of eruption.

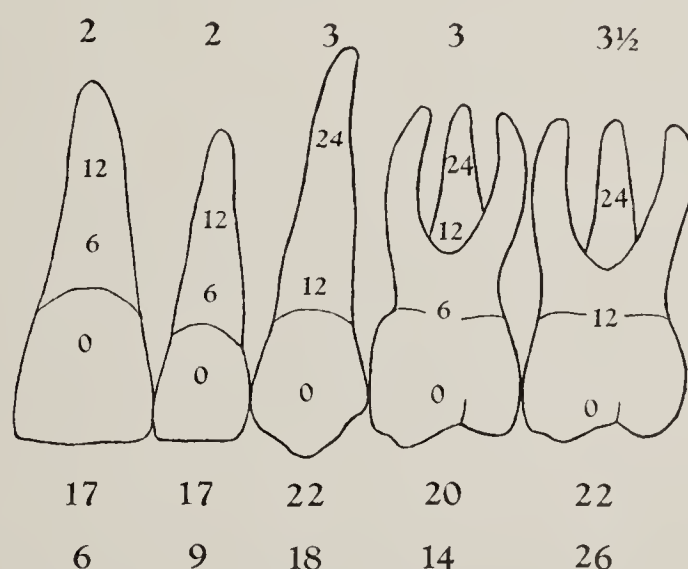


FIG. 10A. Diagram of the temporary teeth, considerably enlarged, representing the progress of their calcification. The figures below each tooth indicate the week of uterine life when calcification begins. 0, placed upon the individual teeth represents the progress of calcification at birth. The figures, 6, 12 and 24, represent, in months, the progress of the calcification of each tooth. The figures above each tooth indicate, in years, the average time of completion of the roots. The figures in the second row, below the teeth, indicate the average age in months, when each tooth erupts. The intention is to represent averages. It must be understood that considerable variations will be found.

ERUPTION. The average time of eruption of the temporary teeth is given in months. The central incisor erupts at six months, the lateral incisor at nine months, the first molar at fourteen months, the cuspid at eighteen months and the second molar at twenty-six months. The time of eruption is probably more variable than the process of calcification. Yet, careful study of cases shows that there is a fair relation between the calcification and the eruption of both the temporary and permanent teeth that is of much value in the management of cases when the history can be obtained. While it does not invariably follow, it is so with sufficient frequency to justify the statement that the late eruption of the temporary teeth usually indicates a corresponding delay in the completion of the roots.

ABSORPTION OF ROOTS. Absorption of the root of the temporary central incisor begins at about the fourth year and is ended at seven. The lateral incisor begins to be absorbed at five years and is ended at eight. The absorption of the root of the first temporary molar is begun at seven and completed at ten years. The second molar has begun to be absorbed at eight and is completed at eleven. The absorption of the cuspid root begins at about eight years and is completed at twelve. See Figure 10B.

There is a difference in time in the absorption of roots of teeth between different individuals. Some will be a year or so early or as much late. Also, the variations for the individual teeth of the person may be considerable. Occasionally the lateral incisors are shed before the centrals, but that is rare. Often they fall away about the same time, or very close together; not six months between. Often again, there will be two years between.

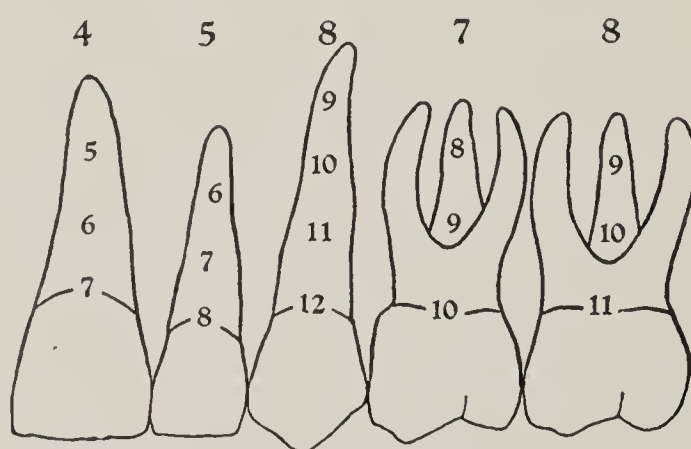


FIG. 10B. Diagram of the temporary teeth considerably enlarged, representing the absorption of the roots. The figure placed over each tooth represents, in years, the average time of the beginning of the absorption of its roots. The figures placed upon the roots of the teeth represent, in years, the progress of the absorption of the roots of the several teeth. Considerable variation from the general average, and also in the order of progress, must be expected.

With the molars it is the same way. Often the second bicuspid will be in place before the first bicuspid, the temporary molars having been shed in this order. The illustration represents only a fair approximation to the average.

ACCIDENTS DURING ABSORPTION OF ROOTS OF THE TEMPORARY TEETH. A number of accidents occur in relation to the absorption of the roots of temporary teeth. First, if there is an alveolar abscess at the root of a temporary tooth and that abscess is continuing in a chronic form, the rule is that absorption of the root will fail. The death of the pulp of the tooth does not interfere with the absorptive process. The question is simply as to the condition of the tissues about the end of the root. The absorption of these roots is a physiological process, and, in order for it to progress properly, the tissues about the root must be in healthy condition. If there is infection, the absorptive process will be defeated.

Occasionally a temporary tooth, the root of which has not been absorbed, will be apparently pushed bodily out of the way by the permanent tooth as it erupts. In other cases, the permanent tooth is deflected from its proper position.

The apical end of the root of a temporary central incisor is occasionally pushed labially, while its crown remains in nearly normal position. Less frequently the same thing may happen to a lateral incisor or a cuspid, but the forms of the teeth replacing these are such that they are more liable to slip to one side and be deflected from their normal positions. The broad cutting edge of the permanent central incisor is more likely to push the root of the temporary tooth labially, causing the end of the root to protrude through the gum and sometimes into the lip. These cases



FIG. 11A.



FIG. 11B.



FIG. 11C.

FIGS. 11A, B, C. Diagrammatic representation of a condition which occasionally occurs, when, because of pathological conditions about the apex of the root of a temporary incisor, absorption of the root fails. Figure 11A. The permanent incisor moves downward and forward to assume its position in the arch and strikes the lingual surface of the root of the temporary tooth near its apex. Figure 11B. In the continued movement of the permanent incisor the apex of the root of the temporary tooth is tipped to the labial and pushed through the gum tissues under the lip. When this is allowed to continue, a condition shown in Figure 11C is liable to occur, in which the apex of the root of the temporary tooth gradually works its way into the lip itself, resulting in suppuration.

are not very frequent, and yet they should be recognized when present. See Figures 11A, 11B and 11C. Whenever the examination of a child, near the age at which the temporary incisors are shed, reveals a sore point under the lip, with some hard substance appearing in the tissues, it should at once be supposed to be the apex of the root of the temporary incisor. A finger of the left hand should be held over the sore point on the gum while the crown of the tooth is grasped between the thumb and finger of the right hand and moved. If the movement is felt by the finger of the left hand, the diagnosis is completed. In the author's practice some years ago, a slight little girl was presented with a sore under the

lip from this cause, which had been neglected until the lip had been cut through and the apex of the root was found in the "running sore" on the skin under the nostril. At the time, the little girl was very thin in flesh, anemic, and had a temperature of 101 degrees. Occasionally the root of the cuspid will be forced labially by the permanent tooth. This root is usually so long that its end will be too high to be forced out under the position where the lip joins the gum, and a part of the labial surface of the root will be exposed through the gum.

In the absorption of the roots of the temporary molars, the crown of the bicuspid will come between these wide-spreading roots, and the roots may be absorbed only near their junction with the crown, and the ends of the roots will be left unabsorbed. These will be found sticking in the alveolar process or gums after the bicuspid has taken its place; sometimes abscesses occur in consequence of this, or occasionally considerable soreness without abscess. Occasionally the unabsorbed portion of the root will remain between the bicuspid and the proximating tooth, expanding the arch and making room for itself. These are usually easily removed if the conditions are recognized. They produce very much less trouble than the roots of incisors. A case may be reported in which a lower first and lower second bicuspid, which were extracted, showed a peculiar growth on the proximal side of the apex of the root of each tooth. Upon examination these growths were found to be the apical half of the roots of a temporary molar, or possibly of the distal root of each temporary molar, that had remained in the jaw and had become attached to the roots of the bicuspids. One of them was slightly movable, and therefore was attached only by the fibers of the peridental membrane, though the attachment was very firm. The other was immovable and was evidently attached by cementum. No history of any difficulty from these retained bits of roots was discoverable.

Occasionally an abscess will occur at the end of the root of a temporary molar before the enamel of the crown of the bicuspid has been completed, and, in that case, the pus may break into the enamel organ and destroy it, or a part of it, so that the enamel of the crown of the bicuspid will not be completed. Then it will erupt with imperfectly formed enamel. Sometimes these injuries closely resemble hypoplasia in appearance, but are readily distinguished from that class of injury by being confined to one or two teeth. It is only occasionally that the alveolar process is injured by an alveolar abscess that has occurred quite early at the root of a temporary tooth.

Not very infrequently the failure of absorption of the roots of an abscessed temporary molar will delay the eruption of a bicuspid; and it is often difficult to determine the cause of this delay satisfactorily without a radiograph. These cases illustrate

the peculiar value of keeping accurate records of cases. A severe alveolar abscess may occur early at the root of a temporary molar; it may be relieved by discharge of the pus, or the removal of the offending tooth, and be forgotten. When the bicuspid takes its place with imperfect enamel, no one knows what has occurred. If there is a record of the prior condition and the treatment, the two incidents become properly connected as cause and effect.

In cases in which the roots of the temporary molar are not absorbed, because of the death of the pulp, and the tooth is to be extracted, a radiograph should be made to learn the relation of the roots to the crown of the bicuspid. It may be enclosed between the roots as though held by a pair of pinchers, and it has occasionally happened that the partially formed bicuspid has been removed with the temporary molar. In all such cases the temporary molar crown should be so divided that each root may be separately removed.

PREMATURE AND DELAYED ABSORPTION. The absorptive process seems to be very fickle in its beginning and in its progress, and there are many cases of variation from the normal. In some of these, absorption seems to be hurried and will be completed before the normal time, so that a temporary tooth will be shed before the permanent tooth has come forward, and the child may be without a tooth for a year or two. In the normal process, when the temporary tooth drops away, the permanent tooth should erupt at once. These cases are not so frequent, however, as delayed absorption. Delayed absorption of roots occurs quite often, so that the coming tooth will be deflected from its position. If the absorption of a root of a lateral incisor or cuspid is delayed, the coming tooth will generally strike its lingual surface and be deflected to the lingual. Deflections to the labial are more likely to occur from other causes, but deflections to the mesial or distal, the tooth making room in the arch for both itself and the retained temporary tooth, are occasionally seen. In case of the bicuspids, the crowns are normally between the spreading roots of the temporary molars and, if the absorption is delayed, the bicuspid is generally held back. In the upper jaw it may escape from between the spreading buccal roots and be deflected to the buccal, so that the tooth will erupt to the buccal of its normal position in the arch. In the lower jaw they may be deflected either to the buccal or to the lingual, but they are more generally simply delayed.

WHEN A SUCCESSIONAL TOOTH IS CONGENITALLY MISSING. In the case where a permanent tooth fails to develop, which occurs occasionally with the lateral incisors, the absorptive process will generally go on and the temporary tooth will be shed, notwithstanding the fact that there is no permanent tooth to take its place. The cuspid tooth, on the other hand, generally remains if there is no permanent tooth coming forward to take its place. If the per-

manent cuspid happens to be deflected from its position from some other cause, or becomes impacted within the bone by taking a wrong direction, the temporary cuspid often remains in its place up to middle life, and in an occasional case it may remain in its position until old age. Any considerable disturbance of such a tooth is likely to hasten the absorptive process and cause the tooth to loosen and fall away, or at least this has been observed in a number of cases where these temporary cuspids, that seemed quite firm in their position, have fallen away soon after a gold foil restoration was made. This has occurred so often as to suggest strongly that a considerable disturbance of the peridental membrane is very liable to start up the absorptive process and cause the loss of the tooth. Therefore, these teeth should be handled very cautiously.

Occasionally temporary molars remain in position, and a bicuspid is deflected mesially or distally and takes its place beside the temporary molar; but, generally, if they are deflected at all, they will be deflected to the buccal in the upper jaw or to either the buccal or lingual in the lower. These temporary teeth do not often remain so late in life as the cuspids, yet one case is recalled in which the patient had one temporary molar still remaining when he died, at about seventy-two. This tooth had done service through all the years, and, of course, where there is a possibility that a temporary tooth will remain, it is important that it have the best possible treatment.

A somewhat singular phenomenon occurs occasionally with temporary teeth that have been retained longer than the usual time of shedding. This is most often seen in the case of the temporary molars when the corresponding bicuspid is congenitally absent, but may occur with the bicuspid present in the jaw. The general rule is that, when these teeth are not shed at the usual time, they are carried occlusally by the growth of the bone, and remain in occlusion. Sometimes, however, the temporary molar, or the two of them, retain their position in the bone, and the growth of the jaws and the movement of the permanent teeth carry the occlusion away from them. These teeth are then often almost overgrown by the gums.

Cases, such as the one just cited, serve to impress the movements of the teeth that are made concurrent with and forming a part of the growth in the lengthening of the face as a part of the change from the child to the adult.

THE RADIOGRAPH is of great value in determining whether or not a retained temporary tooth should be removed. If the radiograph shows the permanent tooth in proper position, and apparently held back, the proper treatment is the extraction of the temporary tooth, with the expectation that the permanent tooth will come forward.

THE PERMANENT TEETH.

CALCIFICATION. The chart, Figure 12, represents the contemporaneous calcification of the permanent teeth in figures placed upon each tooth; a figure for each year during development, indicating the progress of calcification. By following any given figure from tooth to tooth, the particular part of each tooth that has been calcified at the time will be found. When information is desired regarding any particular tooth, the figures placed upon it give the

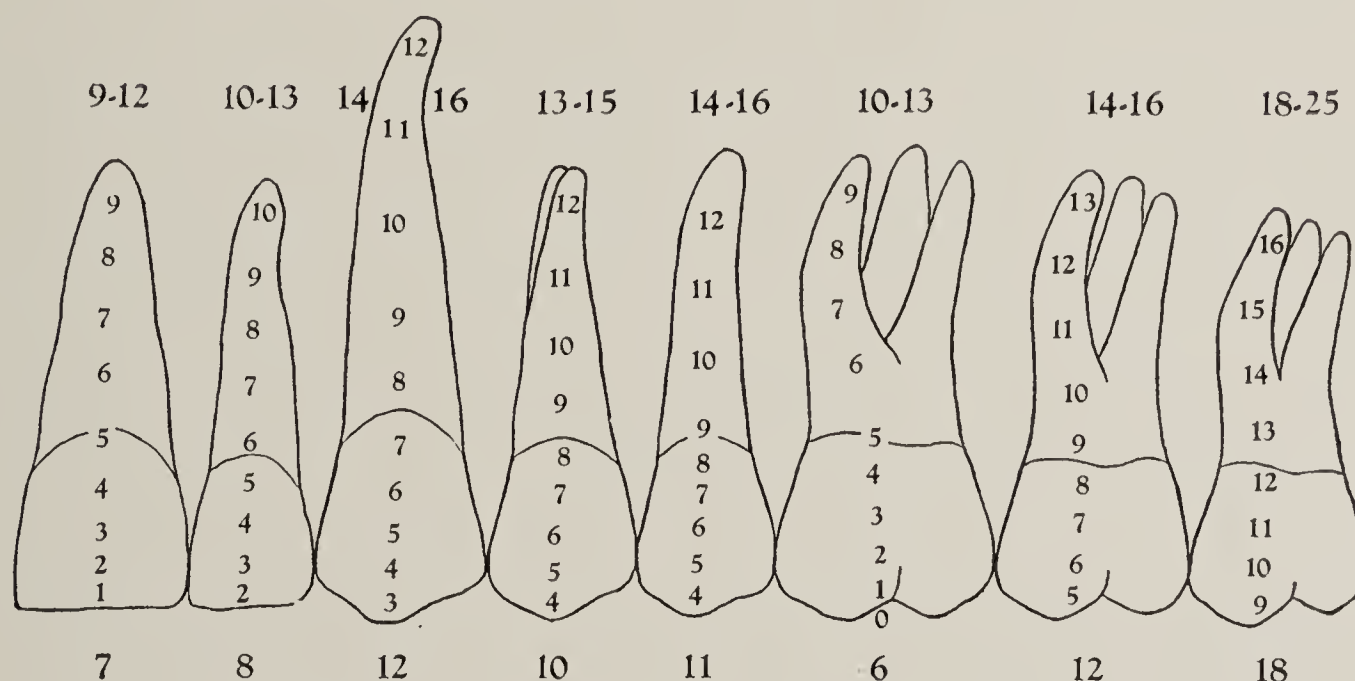


FIG. 12. A diagrammatic representation of the progress of the calcification of the permanent teeth. Below each tooth a figure is placed which represents the average year of the eruption of that tooth. Upon each tooth figures are placed at intervals representing the progress of its calcification to that time, stated in years. The relation of the progress of calcification between the different teeth, or the contemporaneous calcification lines, may be found by following any individual figure from tooth to tooth. The first of the two figures placed above each tooth represents a date at which the apex of the root of that tooth has frequently been found sufficiently narrowed to permit of root filling. The second figure represents the date at which the apex of the root is still occasionally found too widely open for root filling. It must be remembered that in such a diagrammatic representation only an approximation to a general average can be expected. Tolerably wide variations from this average will occur.

growth for each year. The figures placed above the roots of the teeth give the earliest date in the age of the child at which the apical foramen have frequently been found sufficiently narrowed to permit of root filling and the latest date at which they have still been frequently found not sufficiently narrowed. In this the occasional cases of abnormally early teeth and abnormally late teeth are not included.

ERUPTION. The time of the eruption of the permanent teeth is, normally, the same as the shedding of the temporary teeth, but it is specially given in the figures placed below each tooth in Figure 12. The incisors, cuspids and bicuspid are often called the succedaneous or successional teeth, because they take the place of the temporary incisors, cuspids and molars.

There are considerable variations in the time of eruption of

most of the teeth. The upper lateral incisors are very commonly a year, and occasionally two years late; one upper central incisor may erupt a year or more after its mate. Rarely the first molars are from a year to eighteen months late, while one case was observed in which all four first molars were in occlusion soon after the fourth birthday, and two girls, cousins, had these teeth in occlusion when five years of age. The cuspids are often delayed because there is insufficient space for them between the lateral incisor and first bicuspid, which may be due to the early loss of the temporary cuspid. The third molars are often delayed by some interference with their eruption.

IRREGULARITIES OF POSITION DUE TO INTERFERENCE WITH ERUPTION. Irregularities of position of the permanent teeth very commonly result from failures of absorption of the roots of the temporary teeth, as mentioned in the preceding pages. Less frequently the too early loss of a temporary tooth will result in the loss of space for the corresponding permanent tooth. In either case the permanent tooth may be deflected from its normal position. As the care of the temporary teeth receives more attention, irregularities of the permanent teeth will be reduced.

GROWTH OF THE ROOTS. The roots are not completed until several years after the teeth erupt. This is a general rule to which there are some exceptions. The exceptions are almost wholly with teeth that have from some cause been delayed in erupting. Generally the root of the tooth will be completed somewhere near its normal time, even though the eruption of the tooth may be delayed. Sometimes, however, a permanent tooth is delayed in its formation, and there is a corresponding delay in eruption. This seems to occur with the lateral incisors more often than other teeth.

The root of the first molar, erupting at six years, is sufficiently complete at from ten to thirteen years, or from four to seven years after its eruption. The central incisors, erupting at seven, will have the foramen sufficiently closed at about ten to twelve years; three to five years after eruption. If the lateral incisor erupts at eight, the root will be completed at from eleven to twelve; if the first bicuspid comes through at ten, the root should be completed at from thirteen to fourteen; the second bicuspid is usually a little later, both in time of eruption and in the completion of the root. If the cuspid erupts at twelve, the root will usually not be completed before the fifteenth year; if the second molar erupts at twelve, the roots are likely to be completed at fourteen or fifteen.

YEAR REQUIRED TO COMPLETE APICAL FORAMEN. In considering the lengthening of the roots of the teeth, an additional year is usually required after the root has attained its length, for the reduction of the apical foramen. As will be noted in the drawings, Figures 9R and 9S, the root of the lower first molar might appear

the same in a radiograph taken at nine or at ten years, because the growth during the tenth year is confined to the reduction in size of apical foramen, without adding to the length of the root. A good radiograph would generally show the size of the foramen, one improperly exposed would not.

DEVELOPMENT OF THE FIRST MOLAR. The first molar is the slowest tooth in its development. The calcification of this tooth has usually begun at birth. In many examinations in cases of still-birth at term the beginning of calcification of this tooth has been observed. The tooth erupts when the child is six years old, or during the sixth year; occasionally a little before the sixth year, but the average is somewhat later than six-year-old point. Infrequently, much wider variations from the average time occur. The roots of these teeth are rarely completed before the tenth or eleventh year, thus requiring four or five years after eruption for the development of the root, and, in many instances, the length of the root is not complete until six years after eruption, making a total of twelve years from the time of beginning calcification. If these teeth are extracted before the eleventh year the apical foramen will generally not have been closed down to a small opening. The important point is the relation of this closure of the apical foramen to the destruction and removal of the pulp and the filling of the roots.

EXPOSURE OF PULP BEFORE COMPLETION OF ROOT REQUIRES EXTRACTION. It is deplorable to discover an exposed pulp in an important tooth, the root of which is only partially developed, making it impossible to remove the pulp and make a serviceable root filling. In cases in which there is a possibility of the root being sufficiently developed, the effort should be made to treat it, but the patient or the parents should know that it may be necessary to extract the tooth within a few years. In practically all cases in which the apex of the root is not fully formed, including the building of dentin to close the apical opening to a small foramen, it is best to remove the tooth.

THE OCCLUSION OF THE TEETH.

No attempt will be made in this writing to describe in detail the occlusion of the teeth or to analyze the movements of the lower jaw. The object is to sketch briefly the function of the mouth and teeth in the preparation of food for swallowing.

A discussion of all of the factors concerned in mastication would include the maxillae and mandible; the muscles of mastication; the muscles of the cheeks, lips, tongue and palate which control the positions and movements of food; the teeth and their supporting structures and the salivary and other glands of the oral cavity. It seems out of place in this writing to include a discus-

sion of the jaws or the musculature, and in view of the fact that the peridental structures are especially considered in Volume IV, this presentation will be limited fairly closely to the positions and relations of the temporary teeth, the transition period, the positions and relations of the permanent teeth, and the principal movements of the mandible. To this will be added a statement relative to the force that may be exerted by the teeth and the force required to chew various foods.

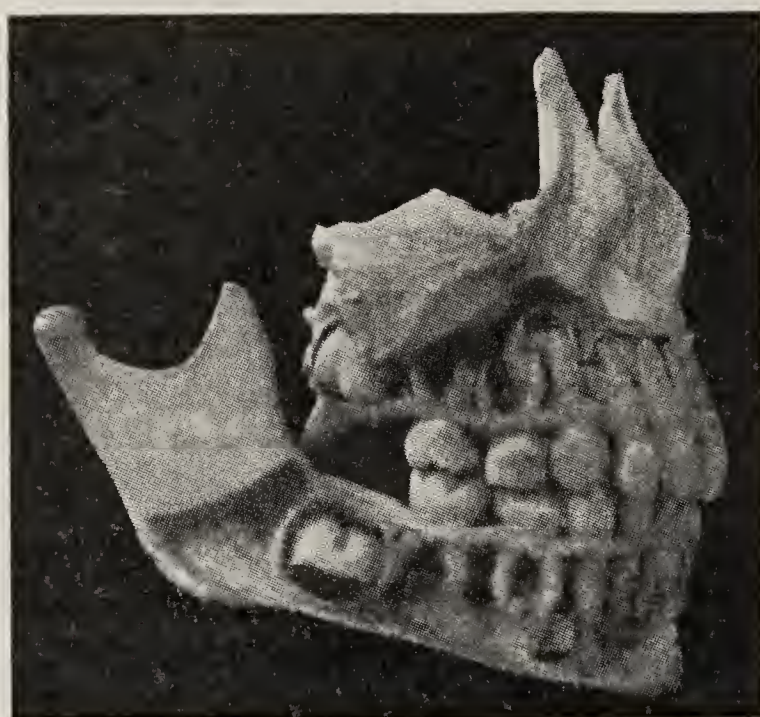


FIGURE 13A. The maxilla and mandible at the end of the second year. The outer alveolar plate has been removed to show the extent of the calcification of the roots of the temporary teeth and of the crowns of the permanent teeth. (Odontological Museum, Royal College of Surgeons of England.)

THE TEMPORARY TEETH.

According to the average schedule, the temporary central incisors erupt at six months, the lateral incisors at nine months, the first molars at fourteen months, the cuspids at eighteen months and the second molars at twenty-four months. As previously stated, there is considerable variation. The eruption of the second molars is the last in a succession of changes which occur in the stomach, oesophagus and mouth that are necessary to the change from a milk diet to food which requires mastication.

The twenty temporary teeth are usually in full occlusion soon after the second birthday. They are more likely to occupy normal relations than the permanent teeth for the reason that neglect of the temporary teeth is one of the principal causes of irregularity of the permanent teeth. As illustrated in Figure 13A, the upper teeth overlap the lowers to the buccal and labial all around the arch, and the total of the mesio-distal diameters of the uppers is correspondingly greater than that of the lowers, although the lower

second molar has the greatest mesio-distal diameter of any of the temporary teeth. The upper incisors and cuspids, particularly, are wider than the corresponding six lower teeth by about 10.4 mm. on the average.

An occlusal view of the lower teeth of half of the arch, with the position of the upper teeth indicated by broken lines, is shown in Figure 13B. It will be noted that the lingual cusps of the upper molars strike in the central fossae of the lowers. This is the posi-



FIG. 13B.



FIG. 13C.

FIG. 13B. Occlusal view of the lower temporary teeth of one side of the arch, with the outlines and cusp positions of the upper teeth superimposed in the positions they occupy when the mouth is closed in the resting position. (Sheldon Friel; *Int. Jnl. Orth., Oral Surg. and Radiog.*, Vol. 13, 1927, p. 326.)

FIG. 13C. Occlusal view of the upper temporary teeth of one side of the arch, with the outlines and cusp positions of the lower teeth superimposed in the positions they occupy when the mouth is closed in the resting position. (Sheldon Friel; *Int. Jnl. Orth., Oral Surg. and Radiog.*, Vol. 13, 1927, p. 325.)

tion in which the jaws are closed in eating meat—the hinged movement of the carnivorous animals. From this position the lower jaw may be moved laterally slightly to the right or left, while the molar teeth remain in occlusion. In this operation the lower teeth slide in contact with the inclined planes of the uppers and the jaws are necessarily opened a little, the amount corresponding to the lateral movement and the angle of the inclined planes of the cusps of the teeth. This movement may be made to either side, also slightly forward and backward, thus providing for the grinding of foods such as are eaten by the herbivorous animals. The lower jaw may also be moved forward so that the edges of the lower incisors come in contact with the edges of the uppers for the purpose of biting or incising food. Thus man is equipped with a masticating apparatus which is a combination of those of the plant and meat eating animals.

Figure 13C is an illustration of the occlusal view of the upper temporary teeth of one side of the arch, with the position of the lower teeth indicated by broken lines.

THE INTERCUSPING OF THE FIRST PERMANENT MOLARS.

The first permanent molars erupt immediately behind the second temporary molars, usually when the child is about six years of age, or about one year before the first of the temporary teeth are shed. See Figure 13D. The proper intercusping of these teeth, upper with lower, on either side of the arch, constitutes the most important single event in the development of the teeth in both its immediate and far reaching effects upon the proper function of the teeth in mastication and in their relation to the growth and development of the lower half of the face, including the most desirable facial expression as an indication of character; also for the effect of normal vigorous mastication on the health of the mouth, with all its implications for the general health.



FIG. 13D. The maxilla and the mandible at the sixth year. The outer alveolar plate has been removed to show the extent of the calcification of the permanent teeth. (Odontological Museum, Royal College of Surgeons of England.)

It will be noted that the first permanent molars are just erupting to the distal of the second temporary molars, while all of the temporary teeth are in position. A comparison of this illustration with Figures 13E, 13F and 13G will show how the first permanent molars maintain their occlusal relation during the transition period.

In the illustration, Figure 13D, the first molars are just erupting, while in Figures 13E, 13F and 13G, they are in occlusion.

The teeth are so formed that those of the lower jaw will intercusp with the teeth of the upper jaw in a certain way, which is very clearly shown from the buccal view in Figure 13E. Also the forms of the teeth are such that if they miss the exact position in which they should occlude a little way only, as they come into occlusion, the tendency is to slide on the slopes of the cusps in such a way as to bring them to the exact relative positions designed — a most beautiful provision of nature for correcting slight deviations that should be closely studied until its meaning is clearly understood. When, however, the teeth are so much out of normal relation at the time that the teeth first make contact in coming into occlusion that the points of the cusps overstep each other ever

so little, the wrong slopes become the moving force directing them into an increased abnormality of position.

If, for example, the mesio-buccal cusp of the upper first molar should strike the point of the mesio-buccal cusp of the lower first molar in such a way that its distal slope instead of its mesial slope slides on that cusp, the upper molar will be moved to the mesial instead of the distal and the abnormality will be increased instead of being corrected. In that case, the upper first molar will be one full cusp width too far forward in its relation to the lower first molar. This error will then be forced upon the other permanent teeth, crowding the front part of the upper arch so that there will be either protrusion of the upper front teeth or irregularity among the bicuspid or cuspid as a result. On the other hand, the case may be reversed. In the relation of these teeth to each other as they approach in coming into occlusion, the lower first molar may be quite a little forward of its normal relation to the upper, but so long as the point of the mesial cusp of the upper strikes ever so little to the mesial of the point of the disto-buccal cusp of the lower first molar, the sliding on the slopes of the cusps will correct the malposition. If, however, the position of the point of the mesio-buccal cusp of the upper first molar should be ever so little to the distal of the point of the disto-buccal cusp of the lower first molar, the sliding will increase the malposition instead of correcting it. The result will be an irregularity or a protrusion of the lower teeth.

The lingual cusps, particularly of the upper molars, play an important part on the same principle mentioned for the buccal cusps. As illustrated in Figure 13J, the disto-lingual cusp should be in a position to the distal of the distal marginal ridge of the lower molar, and the mesio-lingual cusp should be between the mesio-lingual and disto-lingual cusps of the lower molar. In addition to their function of guiding these teeth into their proper mesio-distal relation, these cusps play an equally important rôle in guiding them into proper bucco-lingual relation as well.

The failure of these teeth to intercusp properly causes more irregularities than any other condition. The opportunity for this, however, is usually found in caries of temporary molars by which they have been lost, lost their crowns, or some part of their mesio-distal breadth, which allows one or the other of the first molars to stray too far from its normal position in coming into occlusion with its fellow. Dentists having families of children under their care should discover this particular error in its inception and contrive means for its correction at that time and prevent the impending irregularity. See Volume III, page 90.

SPECIAL FUNCTIONS OF THE FIRST PERMANENT MOLARS.

HOLD THE JAWS IN PROPER RELATION DURING THE TRANSITION PERIOD. The first permanent molars come into position, occluding with each other, just before the shedding of the temporary teeth

begins. Normally, the shedding process begins almost immediately they come into full occlusion. The front part of the arch is soon broken by the shedding of the temporary teeth and these four teeth stand in occlusion, propping the jaws in the position they should occupy during the shedding of the temporary teeth and the eruption of the successional teeth; they also have much to do with the development of the lower half of the face. In the examination of a considerable number of cases, it is found that, with the effect of disease and the irregularities that occur in the absorption of the roots of the temporary teeth and their replacement by the permanent teeth, the support of the jaws is many times almost completely lost but for the presence of the first molars.

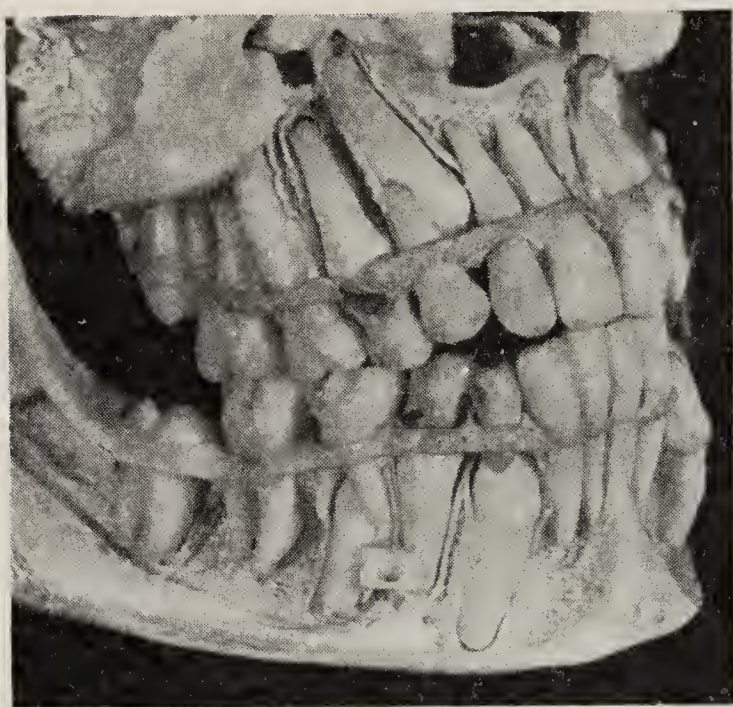


FIG. 13E.



FIG. 13F.

FIG. 13E. The maxilla and mandible at the ninth year. The outer alveolar plate has been removed to show the extent of the absorption of the roots of the remaining temporary teeth and the calcification and positions of the permanent teeth. The first permanent molars are maintaining the relations of the jaws during the transition period. (William Bebb Museum, Northwestern University Dental School.)

FIG. 13F. The maxilla and mandible at the eleventh year. The outer alveolar plate has been removed to show the extent of the calcification of the roots of the permanent teeth. The first permanent molars are maintaining the positions of the jaws until all of the successional teeth have replaced the temporary teeth. (William Bebb Museum, Northwestern University Dental School.)

This important rôle played by the first permanent molars is illustrated in Figures 13E and 13F. Figure 13E is a reproduction of a photograph of the jaws at nine years. It will be noted that the first permanent molars are in proper occlusion, also that all of the permanent incisors are fully erupted. The temporary cuspids have been moved apart by the vertical growth of the face, and the temporary molars have been pushed occlusally by the oncoming bicuspids. The buccal cusps of the second temporary molar strike against the corresponding lower tooth, but has been tipped out of position. There is no occlusion of consequence by the six temporary teeth on this side of the arch; the opposite side is in practically the same condition.

In Figure 13F, illustrating a case at eleven years, the first molars are shown in perfect occlusion with all of the other permanent teeth erupted except the lower second bicuspid (and the third molars), with the second temporary molar still occupying its place and holding the second bicuspid back. The roots of the temporary molar show very little absorption, although they are considerably absorbed on their inner surfaces, immediately about the crown of the bicuspid. In practice, it would be desirable to extract this tooth. Within a few months, if the subject were living, all of the teeth, including the second molars, would be in full occlusion.

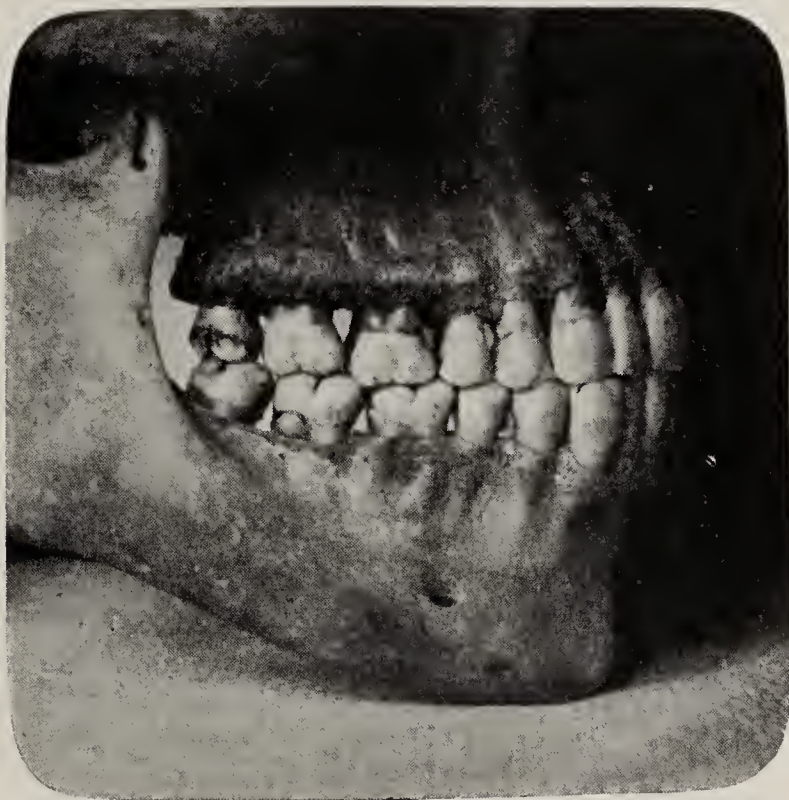


FIG. 13G. The maxilla and mandible of the adult with all the teeth present in normal occlusion.

ASSIST IN DEVELOPMENT OF LOWER HALF OF FACE. Accompanying the shedding process there is a rapid growth of the bones of the jaws and face; particularly the space from the lower orbital ridge to the crest of the alveolar process between the upper teeth is increasing, also the depth of the lower jaw from its lower border to the crest of the alveolar process between the lower teeth. Together these are lengthening the face from above downward. At the same time the whole front of the face is being carried forward, increasing the distance from the ear to the front of the mouth, modeling out that prominence of the features that makes a large part of the difference in the cast of the countenance between the child and the adult. It may be said that the temporary molars act to support the occlusion in the early part of the shedding period, but this is readily shown to be only in part by the many cases in which the bite is raised off from these teeth in the progress of growth of the first molars.

Reference is made to the mouth of a boy almost fifteen years

old, whose temporary molars had not been shed and had not been lifted by the growth of the alveolar processes, which illustrates such a condition. The upper incisors and cuspids overlap the lower in position to slide freely upon each other and could be no support to the occlusion. The jaws are held firmly in position by the four powerful first molars. These, in their usual movement in the lengthening of the face, which is clearly one of their normal functions, had caused the stationary temporary molars to be carried apart about one-third of an inch between their occlusal surfaces. This abnormal case shows plainly the function of the first molars, not alone in holding the jaws in their proper relative positions while the occlusion is broken up in the shedding process, but that they also, by their movement in harmony with the general growth of the face, carry the jaws farther apart and in this way assist in the formation of the features by lengthening the face.

The part which the teeth play in relation to the anterior-posterior dimensions of the lower half of the face is indicated in cases in which, particularly, the first permanent molars are lost before the second molars erupt. The continued prominence of the lower half of the face, in keeping with the growth of the development of the head, is related to the eruption of the first and second molars and to some extent the third molars. The forward movement of the anterior section of the jaw and teeth is very well illustrated in Figures 13A, 13D, 13F and 13G. In Figure 13A, at two years, the mandible has lengthened between the second temporary molar and the ramus to provide space for the first permanent molar. In Figure 13D, at six years, the first molars are about to erupt and more bone has formed to accommodate the second molars, which are in process of formation; also the anterior portions of the jaws, with the temporary teeth, have moved farther forward, increasing the prominence of the chin and upper lip. In Figure 13F, at eleven years, this growth has continued, to provide for the development of the third molars (the formed portions of the crowns have been lost from their crypts in the specimens), and the second molars are almost in occlusion. In Figure 13G, for an adult, the third molars are in full occlusion and the lower half of the face has assumed its full prominence.

The conditions of malocclusion which result from the early loss of one or more of the first permanent molars are mentioned in Vol. III, page 115. In addition, the normal prominence of the chin and upper lip are likely to be lost if any of the molar teeth are lost early, and particularly if the first molars are lost.

DISTORTION OF FEATURES WHEN FIRST MOLARS ARE LOST EARLY. When these teeth are lost early by decay there is apt to be much distortion of the features. This is sufficiently apparent from careful clinical observation, as has been noted by many. From all of these sources of information it seems certain that the temporary molars take no part in the general growth that occurs after the

normal time of the beginning of the absorption of their roots. In most cases they may be carried forward passively with the expansion, or lengthening of the bite, but in many it seems clearly demonstrable that their continued presence later becomes a positive hindrance to development. In these cases at least the normal development would not occur if the first molars had been lost. No measurements have yet been made by which the particular directions of distortion and the amounts have been determined.

Further consideration is given to the evils resulting from the early loss of the first molars in connection with the consideration of caries of these teeth. See Volume III, page 114.

THE PERMANENT TEETH.

As has been stated, the first molars are the first teeth of the permanent set to erupt, at about the age of six; they usually take their places posterior to the second temporary molars with so little disturbance that the child is generally unaware of their presence. As an average, the central incisors erupt at seven, the lateral incisors at eight, the first bicuspid at ten, the second bicuspid at eleven, the second molars and cuspids at twelve, and the third molars at eighteen or twenty.

A buccal view of the permanent dentition in normal occlusion, is presented in Figure 13G. This shows the normal mesio-distal relation of the lower teeth to the upper, with an overlapping of the upper teeth all around the arch. There is a corresponding lingual prominence of the lower teeth. In the mastication of food, it is a function of the tongue and cheeks to place and hold the food between the bicuspid and molars, where the heavy work of mastication is performed. The buccal prominence of the upper teeth and the lingual prominence of the lower teeth prevents one from biting either the cheeks or tongue, as the teeth, which project on the buccal, push the cheek aside as the jaws are closed, and those which project on the lingual push the tongue aside.

An occlusal view of the upper teeth of half of the arch is shown in Figure 13H, with the positions of the lower teeth indicated by broken lines. Figure 13J is a corresponding illustration of the lower teeth, with the positions of the upper teeth indicated by broken lines. The cusp relations of the upper and lower first molars have been discussed. The relations of the cusps of the other teeth to the embrasures and to the opposing sulci are clearly shown. As described for the temporary teeth, these drawings are in the position in which the jaw is closed in chewing meats—with a hinge motion. The lateral and protrusive motions of the lower jaw in grinding and biting are also the same as for the temporary teeth.

In describing the parts of the surfaces of the teeth which are cleaned in mastication and are therefore immune to the beginnings of decay, reference will be again made to these illustrations, which

should be studied with great care to understand particularly the regions of the embrasure surfaces of the teeth that are immune.

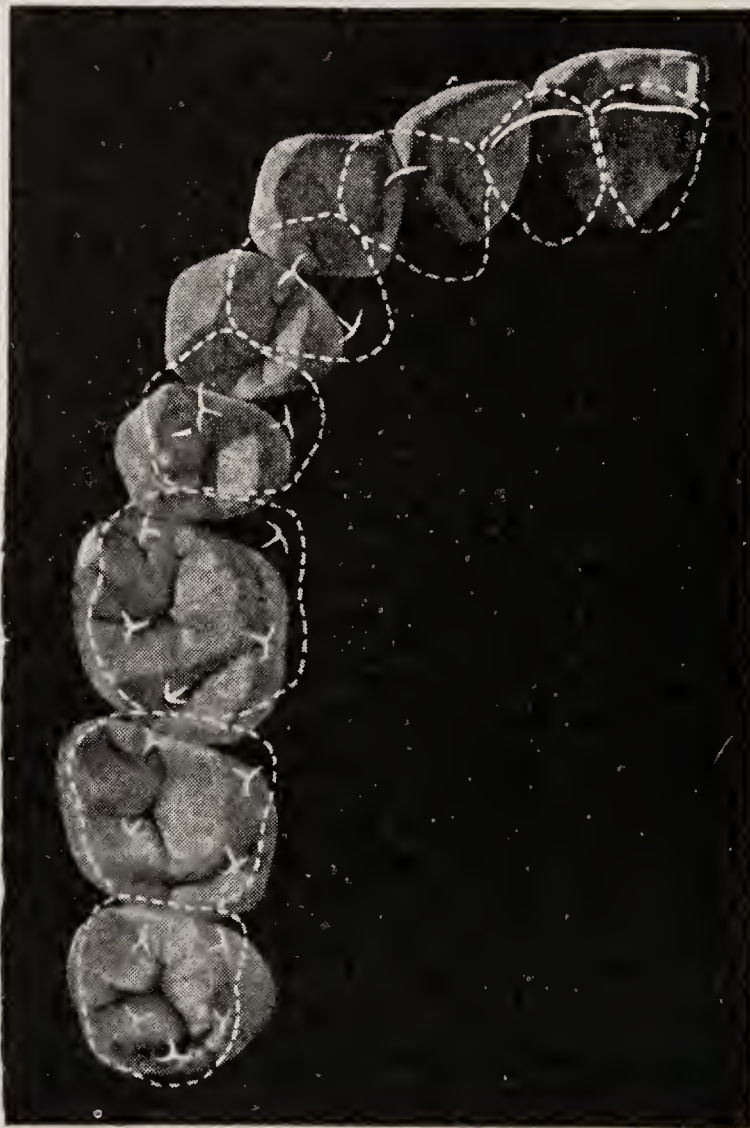


FIG. 13H.

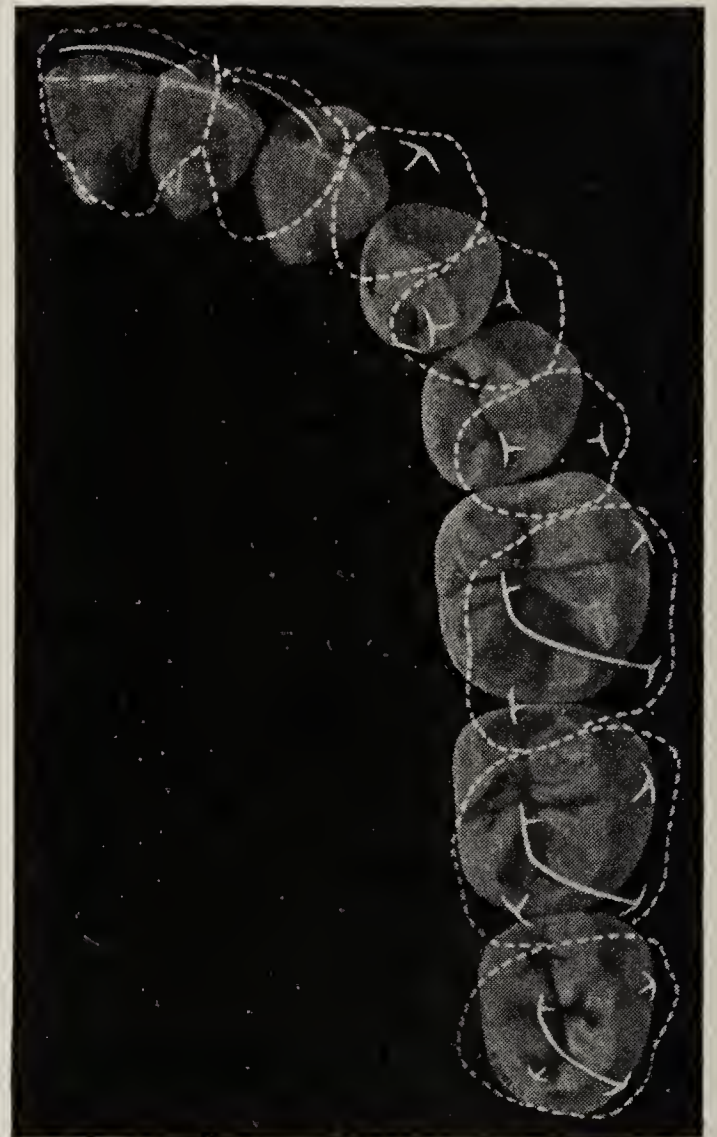


FIG. 13J.

FIG. 13H. Occlusal view of the upper permanent teeth of one side of the arch, with the outlines and cusp positions of the lower teeth superimposed in the positions they occupy when the mouth is closed in the resting position. (Sheldon Friel; *Int. Jnl. Orth., Oral Surg. and Radiog.*, Vol. 13, 1927, p. 334.)

FIG. 13J. Occlusal view of the lower permanent teeth of one side of the arch, with the outlines and cusp positions of the upper teeth superimposed in the positions they occupy when the mouth is closed in the resting position. (Sheldon Friel; *Int. Jnl. Orth., Oral Surg. and Radiog.*, Vol. 13, 1927, p. 335.)

STRENGTH OF THE BITE AND THE FORCE REQUIRED IN THE MASTICATION OF FOOD.

THE STRENGTH OF THE BITE.

The force of the bite, or the pounds force with which the jaws may be closed upon any object, varies greatly among different persons, and is dependent in larger degree upon the condition of the peridental membranes and upon personal habits in the use of the teeth in mastication than upon muscular power. In a tabulation of tests by one thousand young adults, the average force exerted was 171 pounds on the molar teeth, and considerably less on bicuspid and incisors. In this, there was no selection of persons, further than a careful exclusion from the trials of persons

whose teeth were so badly weakened by caries as to cause danger of injury by breakage. Tests of persons of various ages gave the following average figures for the first permanent molars; 8 years, 35 pounds; 12 years, 75 pounds; 18 years, 140 pounds; adults, 175



FIG. 14A. The gnathodynamometer, about two-thirds actual size. C, C. The rubber pads bitten upon in determining the pressure of the teeth. D. Scale of pounds. E. Needle which marks the pounds. In use this needle remains stationary at the highest point reached until it is moved by the fingers.

pounds. A considerable number of young adults have registered 350 pounds. The instrument used for this purpose is the gnathodynamometer — a bite-strength-measure. See Figure 14A.

The amount registered by an individual whose teeth and periodontal membranes are in good condition is greater on the second molars than the first molars and less on the anterior teeth. The strength of the bite on the incisors is on the average, one-half the

number of pounds that may be registered in either first molar region.

The force of the bite of an individual is modified very materially (1) by the use habitually made of the teeth, (2) by infection or injury to the periapical tissues, (3) by disease of the periodontal membranes. Nearly every one who makes trial of his bite on the gnathodynamometer stops because of pain in the periodontal membranes rather than from having reached the full limit of muscular effort. Therefore, modification of the condition of these membranes is prominently brought out. Full and free use of the teeth in mastication, and especially the disposition to use them freely on hard foods, contributes to strength. Any considerable limitation of the use of the teeth, for even a short time, shows in tenderness of the periodontal membranes, and, in cases where the person has fallen into the habit of swallowing food practically without mastication, the power of the bite may be as low as fifty pounds, or even lower. Any considerable pressure in biting causes pain. Very marked cases of this loss of power occasionally occur as a result of disuse of the teeth because of exposure of a pulp, which causes pain that prevents the chewing of food. In such cases, bolting of food is liable to become habitual. This condition is readily recovered from by careful training. In disease affecting the periodontal membranes, the power of the occlusion is rapidly reduced; that is, the muscles of mastication are held from exerting their full power by pain.

The limitation of the force of the occlusion as shown by the gnathodynamometer is a register of the power of resistance by the periodontal membranes and not a register of possible muscular exertion.

The strength of the teeth is ample for all the stress that is brought against them, provided very hard substances are excluded, such as the harder metals and similar things. This is made clear by trials of the strength of freshly extracted teeth. These were cut squarely off at the junction of the middle and gingival third of the crown, so that the occlusal portion would stand solidly on a flat piece of steel, and arranged in a registering dynamometer. Then a steel point with a squared end was applied directly to the cusp and pressure slowly turned on. Another test was made in the same way, except that a slip of hard vulcanized rubber three thirty-seconds of an inch thick was interposed between the steel point and the cusp of the tooth.

In the tests with hard steel directly applied to the points of the cusps of molar teeth, the enamel was checked or pieces were split off at pressures of from 125 to 160 pounds, while in several cases no further damage than a check in the enamel occurred with pressures up to 350 pounds.

In cases in which a piece of vulcanized rubber three thirty-

seconds of an inch thick was placed over the cusp, the cusps were not injured with pressures up to 350 pounds.

The teeth have sufficient strength above that actually required, so that, when considerably weakened by caries, they are still strong enough to withstand the full force of the occlusion on any substance used as food. These may be injured, however, by the accident of catching a small hard substance that might, by accident, be mingled with food. Also the teeth are sometimes, though rarely, broken by catching unawares a lead shot or some such thing so that it wedges between two cusps and splits one off. A cusp that has been weakened by decay may occasionally be split off in a similar way by softer material, such as bread crusts, but it is rather rare that a perfect tooth is broken by any accident in the mastication of food.

FORCE REQUIRED IN THE MASTICATION OF FOOD.

The force required in the mastication of different characters of food is important in this connection. Artificial modes of preparing foods have the tendency to render them softer and softer as so-called improvements are made. In almost every line of food-stuffs, the so-called improvements have rendered them easier of mastication. The instrument used for measuring the force required to masticate various foods is a phagodynamometer — eat-force-measure. See Figure 14B.

About the average force required to chew various meats is as follows:

Cold boiled tongue.....	15 to 20 pounds
Pork chops, loin.....	20 to 25 pounds
Roast pork	30 to 35 pounds
Roast veal	30 to 35 pounds
Boiled corn beef.....	30 to 35 pounds
Mutton chops	30 to 40 pounds
Broiled ham	40 to 60 pounds
Beef steak, tenderloin	35 to 40 pounds
Beef steak, chuck.....	40 to 60 pounds
Beef steak, "boarding house"	60 to 80 pounds

Some of the confections are so hard as to be dangerous to the cusps of teeth that have become weakened by caries.

Crystals of rock candy crushed at.....	30 to 50 pounds
Lemon tablets crushed at.....	50 to 70 pounds
Hard candy (stick), old, crushed at.....	90 to 120 pounds
Hard candy (stick), fresh, crushed at	45 to 60 pounds
Small cinnamon drops crushed at.....	30 to 60 pounds

Some gum drops which were mashed out of shape at twenty to thirty pounds offered great resistance when wedged in between the cusps. Indeed, it often would happen that these could not be completely crushed with less than 250 pounds. Small sticks of licorice offered similar resistance. These and similar articles prove dangerous to the cusps of teeth that are weakened by caries, or to bridges or artificial crowns. Bread crusts are equally dangerous. Indeed, more teeth, that seemed sufficiently strong, have been

broken with bread crusts than with any other one thing. Tests with bread show that it will wedge in between the cusps of the teeth and not be crushed out with a force of 350 pounds.

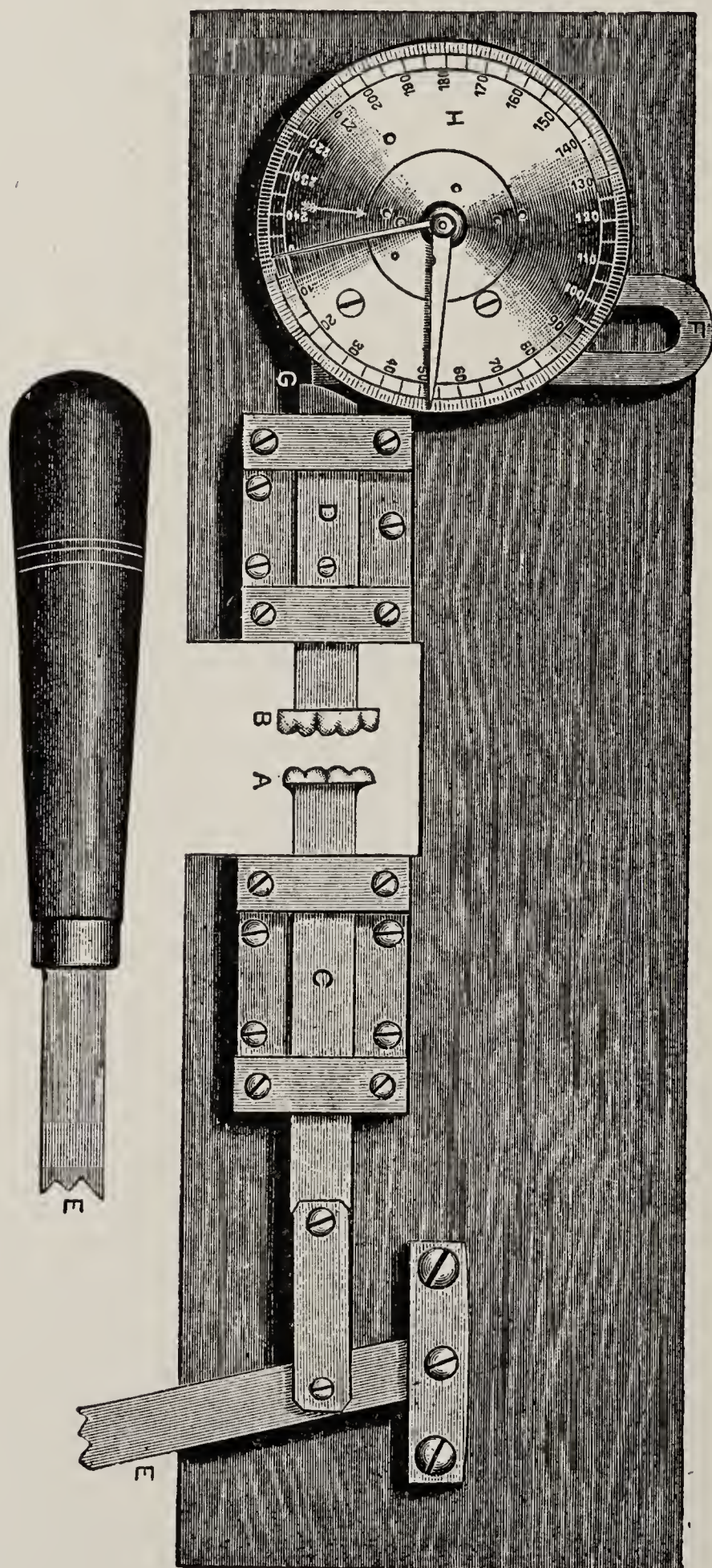


FIG 14B. The phagodynamometer, an instrument designed to measure the force required to crush foods that we eat. A, B. Upper and lower teeth carved of metal. With the lever E, E, these are forced together. The bar D, impinges on the heavy spring F, at G, and the compression of the spring F, moves the hands of the dial H, which registers the pounds force applied. With this the pressure required over the surface of two molar teeth in each jaw may be measured in pounds. It is, in fact, an ordinary spring scale in principle, but arranged for measuring the force of thrusts.

Some persons have objected to these findings because only the direct up-and-down motions were used in tests; claiming that if the lateral or grinding motion were used the crushing would have been accomplished with much less force, and especially that if bread crusts are first wet with water or the saliva they would crush easily. All of this is perfectly correct. Rock candy or lemon drops will melt away in the mouth if the person does not bite upon them, and bread crusts will become soft. But many persons will bite upon them and bite very hard before they have become softened by the saliva. If the grinding motion is used on bread crusts, they will crush easily, but the fact is people are not always careful to use the grinding motion.

In the ordinary chewing of meats, great force is not required, but if some hard substance happens to be in the meat, as a fragment of bone, a shot in game, etc., which is caught between the teeth unawares, the result is a severe jolt upon the teeth. Most persons actually use, habitually, very much more force than seems necessary to crush food. The habit of persons who use their teeth best is to dash them through the food. The jolt on a hard substance is as likely to be received on a restoration as on the surface of the tooth and will give the restoration a severe test. This is liable to occur again and again. Finally, unless the restoration be very strong, it will begin to move. Then it is only a short time until it will be lost. It is for this reason that cavities for restorations which must receive the stress of the occlusion are prepared with a squarely cut horizontal seat, because that will give it the greatest strength. In those cases in which this can not be carried out in full without injury, the nearest possible approach to it should be made. As cavities are formed by caries, the pulpal wall will usually be rounded when the decay is removed; it will be in the form of a hollow sphere. If a cavity is left with this form, it gives the opportunity for the restoration to move, or to roll, when severe stress comes on any particular part of it. Then its usefulness is at an end.

In making restorations in central incisors that have lost an angle, there is usually only about half the stress to contend with as in the molars; yet, in a few instances, as much as two hundred pounds stress has been measured upon these teeth. It is not uncommon for persons to take a hard bread crust between their teeth and put the full force of the muscles of the jaws upon it and then tear it with the hand, putting an additional stress upon them in a direction to exalt the breaking strain.

The cleaning power of vigorous mastication is greater and better than artificial cleaning. It does much for the health of the teeth and the membranes about them and contributes to the general health and vigor of the whole person. Therefore it seems to be the duty of the dentist to cultivate this in his community by careful advice to his patients as he meets them professionally whenever he observes that the teeth are insufficiently used.

DYSTROPHIES OF THE TEETH

THE condition resulting from imperfect, defective, or bad formation of growth constitutes a dystrophy; *dys* — imperfect, defective, bad; *trophy* — growth, development. Therefore, any dystrophy noted must have occurred during the growth of the tissue. A tooth that is misshapen, off color, or otherwise deformed during growth, is in the condition of dystrophy. Acquired deformities, such as erosion, abrasion of the teeth in chewing food, etc., after the teeth have formed, are not dystrophies.

The dystrophies of the teeth consist of imperfections in development due to some disturbance of nutrition during the time of formation or growth. In each class of cases some part of the tissue is either imperfectly developed, or some particular part has failed to develop, or has developed in an erratic manner. Among these dystrophies, there are certain things common to several, such as imperfection of the cementing substance between the enamel rods. In others, the enamel may be wanting or may have an unusual arrangement. In some, defects may be present in all of the hard tissues of the teeth.

These cases are separated in special classes or groups of deformities on the basis of histological studies of the scheme of each. The gross appearance of some of the more frequent of these deformities of the teeth has been well known for many years.

All these deformities thus far seen may be grouped in such a way that each one will be distinguished as a special deformity, and those that are essentially alike may be grouped together. Sections prepared for microscopic observation form the basis of this classification.*

NOMENCLATURE.

Mottled enamel. An endemic deformity, distinguished especially by the absence of the cementing substance between the enamel rods in the outer fourth, more or less, of the enamel, and presenting

* These examinations have convinced the author that the words *atrophy* and *hypoplasia* are no longer desirable. These words have been applied to a specific deformity of the teeth caused by malnutrition, and they have also been used almost continuously in describing abnormal teeth of any and all classes. This they can not properly do, and it seems actually necessary that other words be substituted. The continued use of the word *atrophy* has become a bar to progress, and it will be noticed in this writing that the word *dystrophy* has been substituted as applied to all forms of imperfect development, and other terms are used as descriptive of the several dystrophies. A satisfactory word to replace *hypoplasia* and *atrophy*, as applied to the form of *dystrophy* due to malnutrition, has not been found.

great variety of color. In certain regions of comparatively few square miles, many thousands of persons have this deformity.

Hypoplasia. Atrophy. Contemporaneous accretional dystrophy. A deformity occurring along the lines of accretion, contemporaneously in all teeth in process of development during a period of malnutrition. In this the enamel rods, the cementing substance between the rods, and the dentin are all involved and part of each is either imperfectly formed or wanting.

The enamel whorl. A deformity occurring within the enamel, in which there is an abnormality of direction of the enamel rods, usually associated with a pit in a surface that is normally smooth.

Corrugated teeth. A rare deformity that is characterized by abnormal ridges and grooves of the enamel surface, with scalloping of the dento-enamel junction, and much disarrangement of enamel rods. In each of these scallops there is a disturbance of the direction of the enamel rods. They are thrown into circles and whorls and not infrequently open cavities occur in the tissue.

White spots in the enamel. A deformity observed as a spot which is paper white in the enamel; a form of dystrophy in which the enamel rods are normally formed, but the cementing substance which should occupy the spaces between the rods is missing. These spots are not very frequent, and many of them are passed over without observation. They are of little importance.

White enamel. A deformity similar to the white spots, except that all of the enamel of the teeth is composed of rods without the cementing substance, and the crowns of all of the teeth are pure white. This is a very rare condition.

HISTOLOGICAL CHARACTERISTICS THE BASIS FOR CLASSIFICATION.

To one who has made careful histological studies of the structure of the teeth, these various deformities point to the need for further investigations. Knowledge of the plan on which the teeth are gradually formed from certain points of beginning, makes it possible to recognize one form of dystrophy in which all of the hard structures of the various teeth that were in process of formation at a given time were imperfectly formed, due to an interruption of the normal activities of constructive cells. It is noted that the portions of the teeth formed both before and after the particular time are perfect. This suggests at once a general interruption in the nutritive processes during the period of malformation, and inquiry as to the health of the individual at the age indicated by the malformed part reveals the relationship between the two as cause and effect.

A form of dystrophy is recognized in which there is a failure of the formation of the cementing substance between the enamel

rods in irregular areas, notwithstanding the fact that all other structures of the same teeth, and the cementing substance in other parts of these teeth, formed during the same period, are perfect. Or there may be a partial failure in the development of the enamel rods, as seen in the pits in teeth which are otherwise normal in their formation. Such conditions represent an interruption of the normal activities of certain elements of the formative cells, while cells of the same type close by have functioned properly. These contra-indicate a general systemic disturbance and speak for a purely local interference.

Another form of dystrophy is observed in which the cementing substance between the enamel rods is entirely wanting in all of the teeth of an individual, while every enamel rod is perfectly formed. In this there apparently is a disturbance or lack of activity of an entire group of formative elements, and the cause must be considered as being more general. Lack of a certain necessary stimulus may be imagined, as a result of which the cells which should form the cementing substance have failed to do so. Although no such relationship is known, the situation is comparable to the relationship between certain ductless glands, as the thyroid and suprarenal glands, and other organs and cellular elements. The secretion from these glands in normal quantity is necessary for the proper functional activity of the related organs or cells.

The mottled teeth represent an endemic type of dystrophy, confined to persons living in certain geographic areas. In this there is a failure of the formation of the cementing substance between the enamel rods in the outer third only and in very irregular patches, the cementing substance between other rods being perfect. The fact is well established that these defects occur in the teeth of practically 100 per cent of those persons who live in such areas during the period of enamel formation. If such individuals reside in one of these known geographic areas during the period of formation of a number of the teeth, and elsewhere during the formative period of other teeth, only those which are formed during residence in the area will show the characteristic defects. These present the most difficult problem of all in relating the histologic defect to the cause, since the formation of perfect cementing substance and the lack of formation of this substance are contemporaneous in the individual tooth or several teeth. There is a local failure of cells to function, which is evidently related to some general systemic condition.

These observations indicate that there are separate formative cells for the enamel rods and for the cementing substance between the rods; that under certain conditions both processes are interfered with, while in others there is a failure in the formation of the cementing substance, but not of the rods, and in others a failure of rod formation.

In the following pages the various dystrophies will be described. To get a proper understanding of these conditions in relation to the causes, it is necessary that one have a good knowledge of the histological structure and development of the teeth. There should be in mind for each tooth the average time of beginning formation and the period required for growth to completion. This is necessary to determine the age at which the cause of the particular defect was operating. If it was a condition of malnutrition its effect should be recorded by defects in the portions of all teeth in process of formation at the time. For example, at the age of three about two-thirds of the crown from occlusal to gingival of the first permanent molar has been formed; at the same age only about the incisal third of the central incisor has been formed, usually a little less of the lateral incisor and only the tip of the cuspid. Therefore an illness at the age of three which resulted in a defect of any one of these teeth in the position mentioned should involve all of them. The defect in the cuspid could not be as far away from the incisal edge as in the central incisor, because the central is always in advance of the cuspid in its formation. See Figure 79.

Likewise one should be familiar with the lines of accretion of the enamel — the lines of Retzius — as shown in Figure 80. Each of these lines represents a period of growth of the enamel — a layer, all of which was constructed during a given time. It is without the province of this writing to go into detail in these matters, and the student is referred to the several authoritative works on dental histology.

MOTTLED ENAMEL

By FREDERICK S. MCKAY, D.D.S.

60 ILLUSTRATIONS: FIGURES 15A-60.

THIS lesion attained its first prominence as a problem in dental science, in a certain part of the Rocky Mountain region of the United States through a survey of the public schools, which revealed that it existed in practically the entire native population of that district.

The situation in that form was brought to the attention of Dr. G. V. Black, in 1906 and 1907, and later (1908) the excised crowns of a few affected teeth were sent to him for examination. This was followed in 1909 by a personal examination of several afflicted districts by Dr. Black. The information thus gained, and a description of the histology of the lesion was published by him with the collaboration of the present writer in *Dental Cosmos*, Vol. 58, pp. 129-156, 477-484, 627-644, 781-792, 894-904. 1916.*

From those sources was derived the material included in the chapter on "Mottled Teeth" as published in the prior editions of this work on Operative Dentistry.

That was the beginning of a research which in its major aspects is unique in dental science. There had previously been no description of this lesion in the literature beyond a brief allusion to its occurrence in Italy. Not one fact was known regarding its etiology nor its mode of occurrence, nor was there any knowledge of the extent of its distribution throughout the world. It was later found to be limited to geographical areas whose boundaries are sharply defined, and in those areas, regardless of how widely separated they might be, this lesion presented identical features. It is limited in its occurrence not only to a particular locality but also exclusively to the native population.

Previously, no lesions of the teeth had been recognized, which occurred endemically, and certainly no lesion that is characterized by a geographical distribution. It is this limitation of its occurrence to sharply defined geographical areas that particularly stamps this lesion as being unique. During the investigation some communities

* A brief report was also presented at a meeting of the Panama-Pacific Dental Congress in San Francisco, and published in the *Proceedings*, Vol. 1, p. 25, 20 illustrations; discussion, Vol. 3, p. 3, 1915.

exhibiting an affliction of 100% of the native population were found not more than four or five miles distant from others that were totally immune.

The astounding selectivity of this lesion is another phase of its unique character, affecting as it does only the enamel of such permanent teeth, or such portions of the enamel, as are developed during residence in an afflicted district. Practically without exception all enamel developed within such a district will display this



FIG. 15A.



FIG. 15B.

FIG. 15A. Illinois. A mild case in which areas of grayish mottled enamel are interspersed with more or less normal enamel.

FIG. 15B. Colorado. A pronounced case in which the white mottled areas are more widely distributed throughout the enamel. This individual happens to be a Negro, and is presented as evidence that the lesion is not influenced by racial origin.



FIG. 15C. Arkansas. Practically a uniform involvement of the enamel resulting in the dead white type. The pits and corroded areas on the incisal edges of the left upper lateral incisor and cuspid are referred to in the text.

lesion, and conversely, enamel developed outside such a district will never under any circumstances become mottled, should the individual later become a resident of the district. This feature is illustrated in the case of a given individual who may have developed the enamel or even a portion of the enamel of certain teeth outside an afflicted district, and later establish residence within such a district. The enamel developed prior to the change of residence

will be normal, but all enamel developed subsequently, provided residence within the district is continuous, will be mottled.

Another important fact is that except in extremely rare instances the temporary teeth are not subject to this lesion. In these instances a slight mottling has been observed on the second temporary molars, but the writer has records of not more than two or three cases out of many thousands examined, that presented a fairly typical mottling, mild in degree, involving the entire temporary denture.

This lesion was declared by Dr. Black to be a "new problem in dental pathology" and he named it *mottled enamel*. During the progress of the investigation, as it has proceeded since Dr. Black's first description was published, many facts have been established which tremendously expand the importance of this lesion, making it necessary to revise the chapter, and in so doing it has been thought desirable that the original text be retained in part or suitably adapted.



FIG. 15D. Netherlands. A paper white involvement of the molars.

DESCRIPTION OF THE LESION.

The essential injury in this lesion is the absence of the cementing substance between the enamel rods wholly, or in part, in the outer one-fourth to one-third, more or less, of the enamel. In its typical manifestation this portion of the entire enamel structure, covering all surfaces of all teeth, are affected, although there are differing degrees of severity in different individuals.

According to the degree of completeness of the absence of the cementing substance, the enamel may present a dead *paper white* opaque appearance, or it may be a *mottled* appearance, in which there are white blotches or areas irregularly interspersed with spots of more or less normal enamel. There is none of the translucency of normal enamel. This essential or fundamental phase of the lesion is shown in a series of illustrations, Figures 15A, B, C and D. As will be seen in these illustrations the tooth crowns have not been subject to any macroscopic alterations in their general surface forms as is the case in the contemporaneous accretional

deformities with which mottled enamel is frequently confused. This will be referred to later.

CORROSION OF THE ENAMEL

In mottled enamel, the usual glaze of the surface is complete, that is, Nasmyth's Membrane, which covers the outer ends of the enamel rods, is normal. A sharp exploring tine will glide over the surface without catching, the same as over normal enamel. There are, however, grosser manifestations of this lesion, involving actual damage to the continuity of the tooth surface for which there had



FIG. 15E.

FIG. 15E. Idaho. Corroded spots on upper and lower central incisors.



FIG. 15F.

FIG. 15F. Idaho. Corroded areas on upper central incisors and pits on various other teeth.



FIG. 15G.

FIG. 15G. Idaho. Corroded areas on upper central incisors.



FIG. 15H.

FIG. 15H. Illinois. A typical corroded involvement of the tips of the cusps shown in the right upper cuspid and first bicuspid.

previously been no descriptive term. In an article published in *Dental Cosmos** the present writer in describing it applied the term "corrosion of the enamel", which while not strictly accurate in its implication seems to be acceptable. That term, therefore, will be used throughout this chapter. Some common examples of a milder phase of this are shown in Figures 15E, F, G and H. As a rule the

* McKay, Frederick S. "The Establishment of a Definite Relation Between Enamel That Is Defective in Its Structure, as Mottled Enamel, and the Liability to Decay." *Dental Cosmos*, 1929, Vol. 71, p. 747; also *Pacific Dental Gazette*, Vol. 37, p. 599.

pits and corroded areas are more pronounced on the labial surfaces and may be entirely absent on the lingual surfaces.

The individual whose teeth are shown in Figures 16A and B, was born and reared in an endemic district. At the time these teeth were extracted this person had attained adult life but had retained the temporary molar shown at the left of Figure 16A, the enamel of which was normal, supporting the observation that the temporary teeth are not affected. This observation is further illustrated in Figure 16C which shows the remarkable difference between the permanent incisors above and below, which are typically mottled,

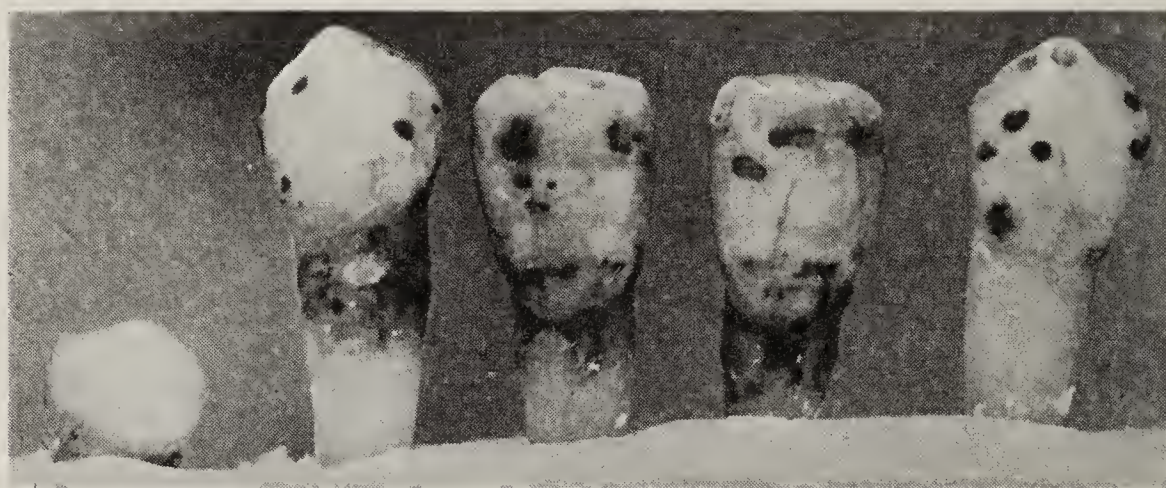


FIG. 16A. Colorado. This magnification gives a good idea of the pits on labial surfaces. The temporary molar at the left, with normal enamel, is from the mouth of the same individual.



FIG. 16B. Colorado. Lingual surfaces of the same incisors and cuspids as in Fig. 16A, with almost complete absence of pits.

and the temporary teeth, which are normal. This person was a native of an endemic district. In illustrating the more severe cor-rosions in Figures 17A to F inclusive, no particular descriptive text is necessary except to say that Figures 17C, D, E and F show the most extreme damage.



FIG. 16C. Colorado. Mottled upper and lower permanent incisors with normal temporary teeth.

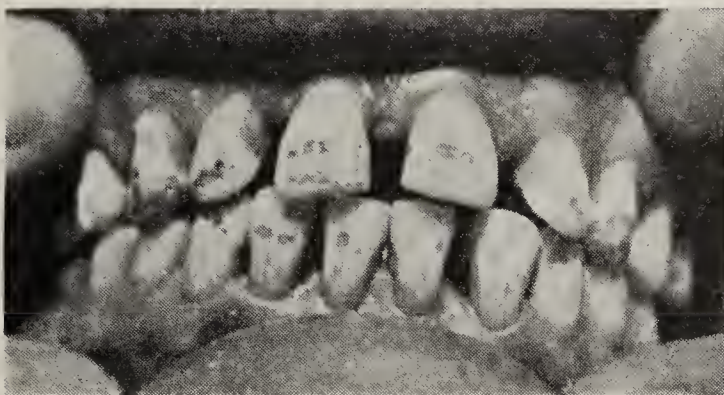


FIG. 17A.

FIG. 17A. Barbados. Corrosion of the enamel.



FIG. 17B.

FIG. 17B. Arkansas. Corrosion of the enamel.



FIG. 17C.

FIG. 17C. Arkansas. Corrosion of the enamel.



FIG. 17D.

FIG. 17D. Arkansas. Corrosion of the enamel.

Associated with the phases of this lesion that we have been describing, there is found in many cases a brown discoloration, of all varieties of shade, lodged within the enamel structure. This phase has commonly been referred to as the *brown stain* and constitutes the most distressing phase of the unsightliness in the appearance of this lesion. There is every degree conceivable in the co-mingling of color from light yellow to brown and even black.

The distribution of this coloring matter is most irregular and erratic as will be seen in illustrations that are to follow, but an amazing peculiarity is that it is confined to the labial surfaces of the upper incisors and cuspids. In the experience of the writer it has never been observed to cross the incisal edges of these teeth and involve the lingual surface, even when the incisal portion of the labial surface was deeply stained. Neither has it been observed to occur on the lower incisors, which fact is well demonstrated by the



FIG. 17E.

FIG. 17E. Arkansas. Corrosion of the enamel.

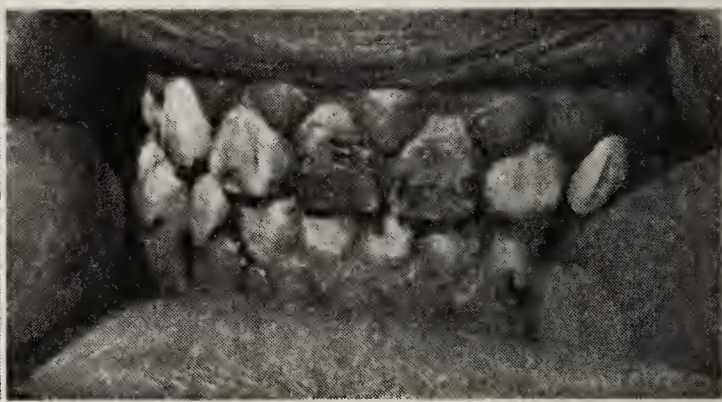


FIG. 17F.

FIG. 17F. Pima Indian, Arizona. Corrosion.



FIG. 18.

FIG. 18. Colorado. Paper white enamel with slight brown stain on upper central incisors.



FIG. 19.

FIG. 19. Idaho. Fairly symmetrical distribution of stain.

illustrations that follow. Cases have been observed, however, in which the entire denture, including the third molars, has been of a smoky color evenly distributed through the enamel. Quite often the discoloration will occupy a considerable portion of the central incisors, perhaps across the middle third, a lesser portion of the lateral incisors, more toward the incisal edge, and just the tips of the cuspids, and in a fairly symmetrical manner on the teeth of both sides of the mouth. The appearance of the *paper white* teeth

is glaring when exposed to view, but not comparable in its disfigurement with the cases where the brown stain has occurred.

Almost every conceivable phase of the discoloration is shown in Figures 18 to 24 inclusive. It is to be emphasized that most of these teeth are perfectly smooth over the stained areas; that is, the enamel surfaces are covered with the original Nasmyth's membrane intact. It is also to be noted that the lower teeth are free from the stain as pointed out previously in the text.



FIG. 20.

FIG. 20. Illinois. Symmetrical stain on upper incisors.



FIG. 21.

FIG. 21. Colorado. Fairly symmetrical stain on upper central incisors and occlusal edges of lateral incisors.



FIG. 22.

FIG. 22. Colorado. Paper white with stain symmetrically located on upper central incisors.



FIG. 23.

FIG. 23. Netherlands. A very dark discoloration. Extracted teeth mounted in plaster.

The series of illustrations, Figures 25 to 28, show stained cases in which there has also been corrosion in varying degrees of severity.

There has been a constant misconception in the understanding and use of the term *mottled*, hence it is important to point out that this term applies exclusively to the grayish white or blotched appearance of the enamel, and even includes the dead white phase, and does not refer to the brown discoloration nor the corroded types.

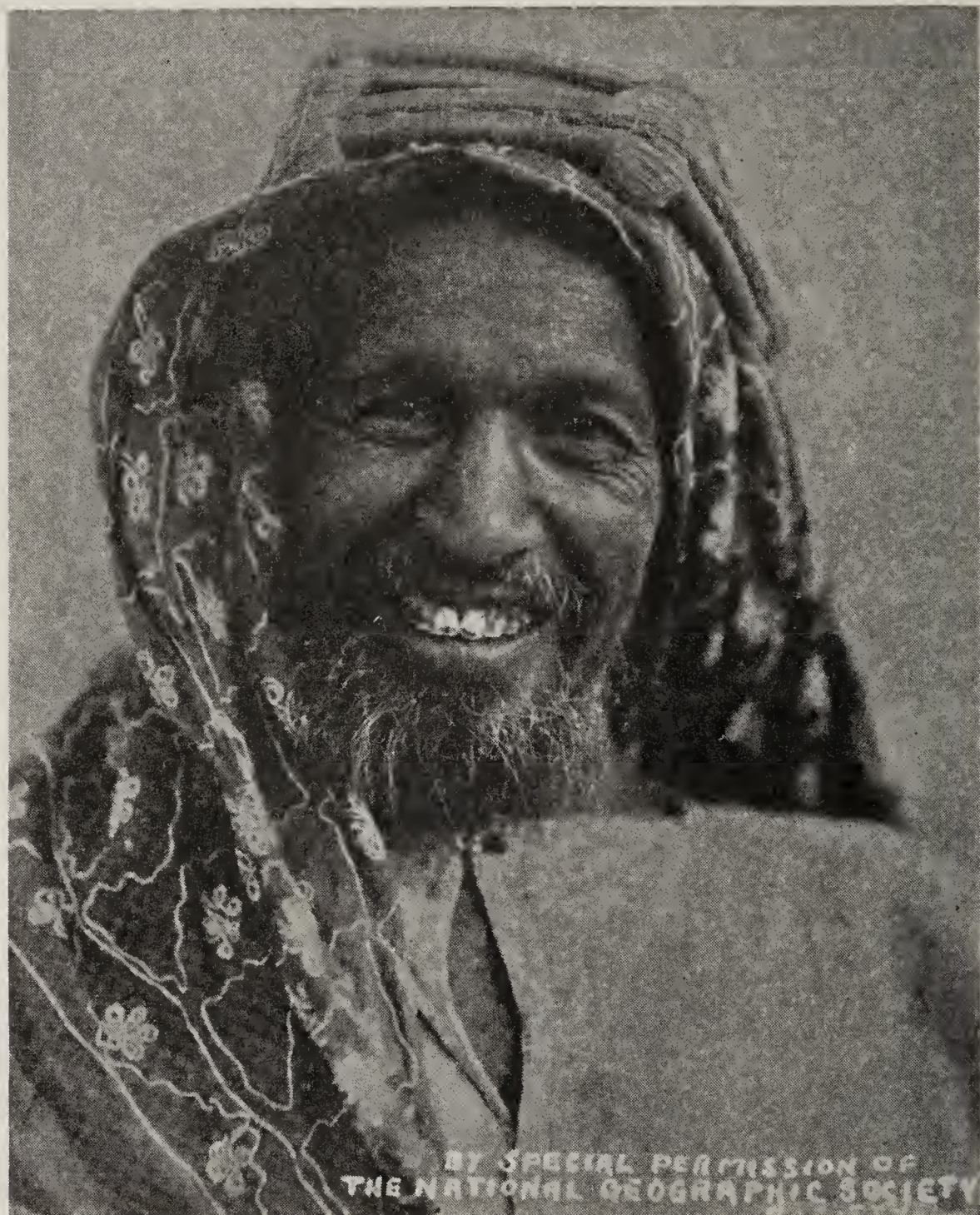


FIG. 24. Sheik of the Desert. Sinai Peninsula, Northern Africa. An Arab. Photograph by courtesy of National Geographic Society. Nothing is known of the etiology of this case.

COLORING MATTER: BROWNIN.

In Black's studies of the enamel lesions in general, he had found what he believed to be this same coloring matter in most kinds of malformations of enamel. Especially is it abundant in the contemporaneous accretional deformities. Its distribution in mottled enamel has been described in the preceding section of this chapter. In the enamel whorl a discoloration is found deep in the enamel sometimes actually lying against the dento-enamel junction, making a very dark spot in a section, while the surface of the tooth has no appearance of discoloration. The coloring matter is the same material, and its reaction to dissolving agents is the same in all of these various conditions. When dissolved out such teeth are white. In an effort to find a suitable name for this coloring matter

for use in dentistry, Black used the word *brownin*, which seems in good form as a nomenclature word.

Its composition is as yet not definitely known, although from a chemical analysis of a central incisor, which was extracted because of an accident, it was stated to be manganese, which determination, however, lacks complete confirmation. This was reported in *Dental Cosmos*, Vol. 69, p. 736.



FIG. 25.

FIG. 25. Arkansas. Stains on upper central incisors with corrosion on left lateral incisor and cuspid.



FIG. 26.

FIG. 26. Arkansas. Marked corrosion on upper incisors with stain on incisal portion of central incisors.

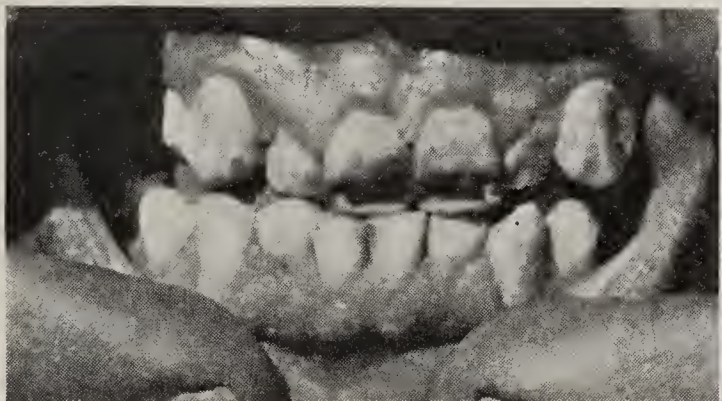


FIG. 27.

FIG. 27. Pima Indian, Arizona. Stain on upper central incisors. The enamel surfaces of the rest of the upper teeth are generally rough and irregular, but not definitely corroded.

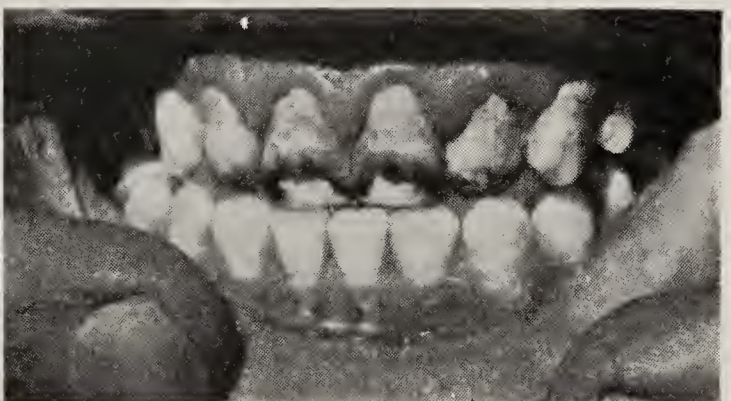


FIG. 28.

FIG. 28. Pima Indian, Arizona. The same general description as applied to Fig. 27.

In the earlier studies it was thought that the discoloration occurred before the teeth erupted. The idea that this colored material was laid into, or gained access to the damaged area of the enamel during the calcification process, was supported by the presence of deep-seated discolorations in other forms of enamel dystrophy, and also by the general intactness of the outer enamel surface. Extended observations in endemic districts, however, have established that these stained areas are never apparent on newly erupted teeth, but that they gradually appear after eruption. Whether this is due to a gradual chemical alteration of a substance already lodged within the enamel, by means of which process a pigmentation is

developed, or whether due to an extraneous substance gaining access to the enamel after eruption, is as yet not determined. Its presence on the labial surfaces of the upper incisors as illustrated, gives some credence to the theory that it may be due to the action of light. This theory is in a measure contradicted by those cases referred to in which there was a general smoky appearance of the entire denture.

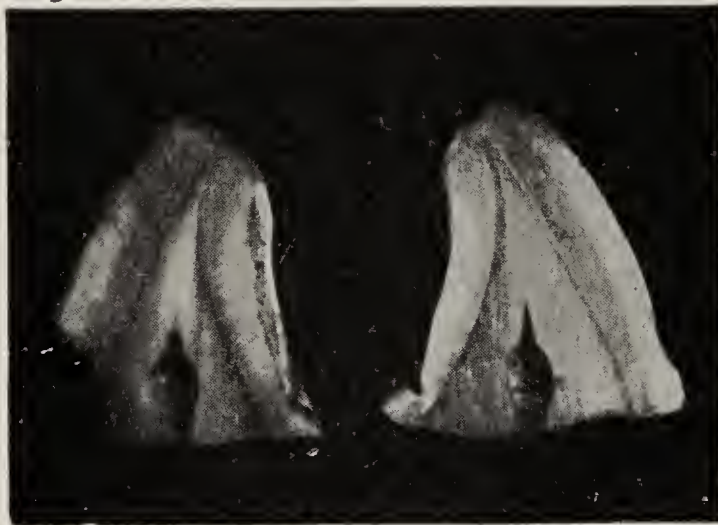


FIG. 29.

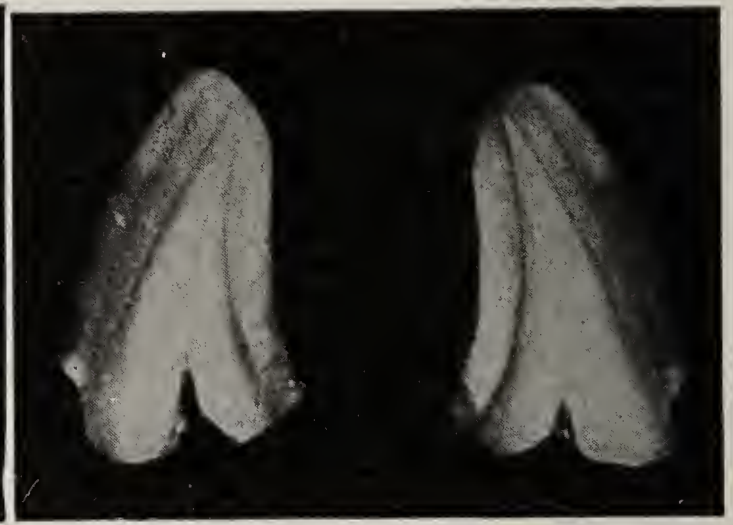


FIG. 30.

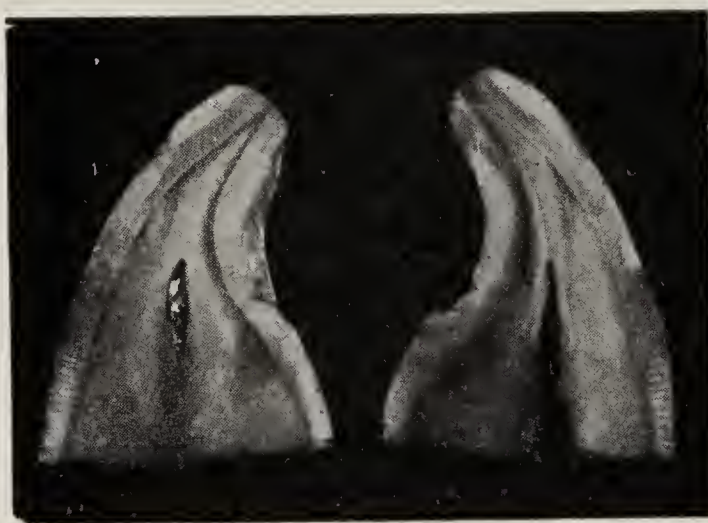


FIG. 31.

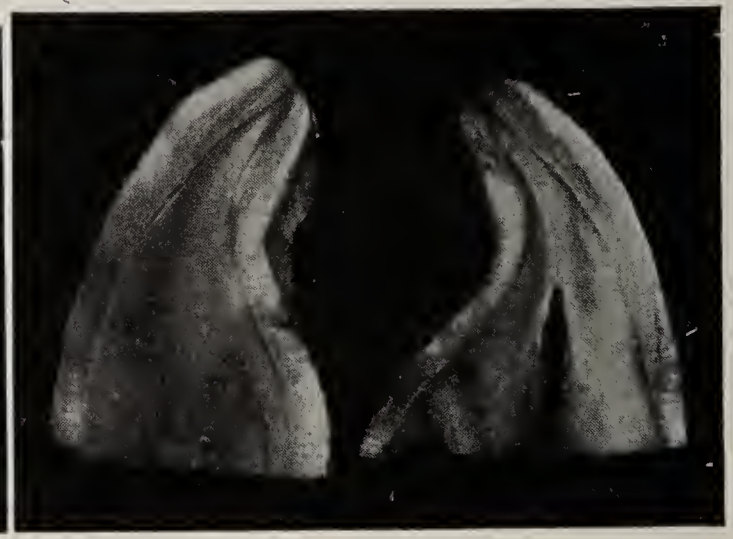


FIG. 32.

FIGS. 29, 30, 31, 32. These figures represent teeth that had been split longitudinally and very perfectly in line. They had been cut off for the purpose of placing artificial crowns, and did not show the full length of the crowns. Figures 29, 30 and 31 are central incisors, and Figure 32 is a lateral incisor. The surface of the enamel is a different color from the inner portion. This may be seen also on the lingual surface, but it is not so prominent. This is the injured part of the tooth in mottled enamel. The thickness of the injury can be made out by careful examination of the figures.

It is a significant fact that normal enamel, in vivo, is rarely, if ever permeated by a stain. The presence of coloring matter within such enamel structure is associated with an injury to this tissue which has been inflicted during its developmental period, and its occurrence in mottled enamel is made possible because of the existing structural defect, about to be described.

HISTOLOGICAL CHARACTERISTICS.

The prior editions of this work have contained a description of the original histological work on mottled enamel by Black which is given below as it there appeared, the quotation marks indicating the original text. The teeth referred to were sent to him from an endemic district in Colorado. See Figures 29 to 35.

"The mottled teeth which I received were split labio-lingually through their centers longitudinally when they came to me. Many of the crowns were incomplete in that they had been cut from the roots some little distance from the gingival line. I ground the broken surfaces flat, polished them, and photographed them mounted in the pairs from each tooth. These were photographed by reflected light with enlargements of from six to eight diameters. (See Figures 29, 30, 31, 32.) The material did not give very brilliant pictures, but the outlines of the imperfectly developed enamel were shown.



FIG. 33. Photomicrograph of a section of moderately dark enamel from a mottled tooth. There is no cementing substance between the enamel rods, the space being filled with brownin.

"Sections were then ground for microscopic study. They presented a very considerable variety of injuries. The teeth were all from young persons, and were practically unworn except a few on the incisal edge. The enamel was normal in its outline form and normal in thickness in all of the specimens, but not normal in color. The group presented, as I found later by personal examination of many children, a series of bad cases of mottling. Some portions of the enamel were perfectly normal, both in color and histological development, in the majority of the specimens. A number of them were of a very dark brown color over a considerable portion of the



FIG. 34. The distribution of the brownin between the enamel rods is shown at A, B, C, D.

labial surfaces, shading from the brown areas through varying shades of yellow, to opaque paper-white, and from this into the normal enamel color. All of the abnormal areas showed the same lack of development of the cementing substance which usually binds the enamel rods together. The degree of this injury varied in the different teeth and in the different parts of the crowns of individual teeth. Later, in examining the children, I saw many teeth that were much darker in color than those I had for cutting. This, however, was only a matter of degree of injury without difference in kind.



FIG. 35. In examining this figure the reader should have fixed in his mind the following: the substance of the perfectly developed enamel of the tooth has not been successfully stained; being a solid, it will absorb nothing into itself, therefore any staining in the substance of the enamel is the result of imperfect development which creates openings which will receive a stain.

The photomicrograph is of a portion of the crown of an incisor tooth from which all traces of color had been dissolved out, making the tooth very white. The piece was placed in alcohol for several days and then transferred to a solution of shellac which had been tinted with gentian violet. After remaining in that for two weeks it was mounted upon a cover-glass and dried for grinding, and a section was cut.

It will be seen that the labial surface of the tooth — the portion most injured by failure of the cementing substance between the enamel rods is black. The lingual surface is very different; the injury has been much milder than on the labial surface, and areas of white run through the dark areas, also the brownish shows in long lines instead of making a full brown. Only a portion of the cementing substance between the enamel rods has failed.

“In all of the specimens the enamel rods were well formed * throughout; in the imperfect areas the enamel rods seemed as regular and perfect in form as in the areas in which the cementing substance between them was normal. In areas in which the dif-

* Later histological studies by the late J. Leon Williams present evidence that the rods themselves were damaged by a reduction in general dimensions and distortion of shape. *Journal of Dental Research*, 1923, Vol. 5, p. 117.

ficulty was simply a lack of the cementing substance which should be between the enamel rods, the spaces were empty, or filled with air. Such areas were opaque paper-white because of the presence of air between the enamel rods.

"In the dark-colored areas the brownin was found to be in the spaces between the enamel rods. The enamel rods themselves were as perfect and presented the same cross markings as in normal teeth, but they often made very dark photomicrographs. (See Figure 33.)"

"The lines of accretion in the growth of the enamel were about the same as in normal enamel. It was particularly notable that the lines and depths of the abnormal condition had no reference whatever to the lines of accretion or growth in the formation of the enamel, thus showing a remarkable difference from the contemporaneous accretional deformities of the enamel, in which the lines of accretion in the growth of the tooth are very closely followed."

It is important to emphasize in this way the dissimilarity between mottled enamel and hypoplasia. A few cases have been observed in which these two distinct lesions have been combined in the same individual.

"In the illustration, Figure 33, it will be noticed that there are areas or lines of brownin distributed deeply in the enamel. The yellow shades of stain seem to be caused by brownin within the substance of the enamel which is covered by normal enamel. In this case the modification of color is caused by partial showing of the brown through the translucent covering. In other cases the yellow color appears to be caused by minute areas of brownin too small for the naked eye to distinguish as separate, and the mingling of this with the translucent white gives the yellow shades. In my sections I found no yellow colors whatever.

"Distribution of brownin in the enamel as a whole is extremely irregular. In the darkest areas I found in the teeth furnished me, it was difficult to make a photomicrograph that would show all of the spaces between the rods filled. Many of them are empty. This character of the deposit is quite well shown in Figures 34 and 35, which were made with low enough power to show the whole of the incisal portion of the teeth.

"The sections cut from teeth that have been mounted for grinding in very light-colored shellac show plainly that the limit of the imperfect enamel is not a sharp line, but that some of the spaces are open between the enamel rods much deeper than others. This causes the color produced by brownin to thin out into the perfect enamel.

"There are also in this enamel many places where the color seems to be about normal, in which small groups of spaces between the enamel rods are filled with brownin. This is very sharply brought out in some of the photomicrographs. Many of these

islands of color are so small as to escape observation with the naked eye, but come out prominently with the medium powers of the microscope."

Up to the time of writing no evidence is at hand to indicate that the dentin underlying the mottled enamel has been damaged or its normal calcification interfered with.

MODE OF OCCURRENCE.

In describing a region or a community as endemic to mottled enamel we understand that the lesion is in constant occurrence in practically the entire native population. To maintain a continuous residence in such a community during the period of calcification of the permanent teeth will result in mottled enamel of all teeth. In other words all enamel grown or calcified in an endemic district will display this lesion when the teeth are erupted. The fact of birth in an endemic region is not the qualifying factor; a child whose residence may be established there during the first year of life is included as a "native," for the reason that calcification of the permanent teeth is almost, if not completely, post natal.

All enamel that has calcified prior to a change of residence from a non-endemic into an endemic district will remain normal, and conversely, all enamel calcified subsequent to the removal into the endemic district will be mottled. All enamel that has become mottled by reason of residence in an endemic district will remain so permanently, even should there be a change of residence later into a non-endemic district, and conversely, all enamel that undergoes calcification in a non-endemic district subsequent to a change of residence from an endemic district will be normal. In other words, no normal enamel ever becomes mottled, nor does mottled enamel ever become normal, regardless of any change of residence.

As often observed, the normal and the mottled enamel, on certain teeth of an individual, will be separated by a sharp line of demarcation, offering a means of determining the approximate age attained at the time of a change of residence, that is almost mathematical in its accuracy. The reason for this is important not alone in connection with this subject, but as related to dental science in general, offering substantial support to the contention that enamel, after its calcification has been completed, is not thereafter subject to physiological influences.

Black says: "Any departure from the normal in the enamel, the dentin, or the form of the teeth, from errors in development, must occur while the teeth are growing. This must be differentiated sharply from deformities that are acquired after the teeth have grown. The tissues of the teeth are not changed in any wise by physiological processes after they are once formed." The injury thus inflicted during the developmental period is permanent and remains static through life, that is, the enamel neither improves

through natural means, nor does it undergo any further deterioration in structure of similar nature, even through continued residence in an endemic district. The amount of damage to the structure that has been inflicted prior to eruption is complete and final, and, limited by the very nature of the enamel structure, anything simulating a repair process through natural means is not possible.

"The deciduous teeth, with rarest exceptions as previously stated, are always normal in this respect. The permanent teeth are naturally divisible into three groups. The first group includes the first molars, the incisors and the cuspids. The enamel of this group is growing during the first five years of the child's life, excepting that the cuspids frequently continue to the seventh year. The second group includes the bicuspid and second molars. The enamel of this group begins growing at from five to six years, and is completed at from nine to eleven. The third group includes the third molars only. The enamel of these is growing ordinarily from the tenth to the fourteenth or fifteenth year, but presenting considerable variation of the time of completion of the growth. This is an approximate statement.

"It occurs, therefore, that if the child is not in the locality of endemic mottled enamel during the time of the growth of the enamel of any one of these groups of teeth, that group will not be marked. Or if a child is in the locality only during the time of the growth of one, and elsewhere the rest of the time, only that one group of teeth will be marked. It follows that if a child born in the locality is removed and lives elsewhere for the first five years, the first molars, incisors and cuspids will be normal. If the child returns to the locality at the end of the five years and continues to live there, the other two groups of teeth will be mottled, and if a child is not brought into an endemic district until the age of twelve or thereabouts only the third molars will be mottled."

With relation to the time in the life of the child in which the enamel in different groups of teeth is growing, having reference only to the permanent teeth, Black can be quoted further as follows:

"Having lived a part of the time in this area does not seem to carry with it a continuance of the injury after removal. Neither does living elsewhere during the growth of the enamel of the incisors, and then coming into the endemic area, prevent the injury to the teeth which have yet to grow their enamel."

There are border line cases among natives of endemic districts, that is, cases in which the injury has been so slight as to make diagnosis difficult, that are hard to account for. There are variations also in the degree of the intensity of the injury in individuals of the same family who naturally have been subject to the same environment and family habits. This is true not only in the severity of the fundamental defect, but in the extent and intensity of the stain as well. This can be illustrated by a glance at Figures 42

and 43, which show the teeth of twin boys born and raised on a South Dakota ranch.

It is a matter of somewhat common observation in endemic districts that native children who have been taken out of the district for a considerable portion, say from two to four months, of each year, from the ages of one to five, will escape the damage in large part, if not wholly, particularly on the incisor and the cuspid group. At the present stage of our knowledge of this subject it is not possible to account for these differences among natives of continuous residence in afflicted districts, although it has been hinted that dietary differences might be accountable.

ETIOLOGY.

Lesions of the enamel are limited to two general classifications:

1. Those produced through external influences after eruption, such as abrasions and erosions, and the decalcification in the first stage of dental caries.

2. Those produced through interference with the normal process of calcification during the developmental period.

These are usually referred to as *hypoplasias*, but more correctly as dystrophies, which term signifies a disturbance of nutrition, which is exactly what happens. The general use of the term *hypoplasia* is subject to the criticism that it is applied to one of a group of conditions of dystrophy. In all of these conditions the enamel is hypoplastic, be it the contemporaneous accretional deformity or mottled enamel, yet totally dissimilar both in appearance and in etiology.

Before the recognition of mottled enamel as a phase of enamel pathology, it was generally considered that the so-called hypoplasia, or atrophy, surpassed all others in the number of teeth involved, but with the wide distribution of mottled enamel now known to exist, this latter condition can probably be considered as the most frequently occurring dystrophy of the enamel. Supporting this assertion it will be recalled that in a case of the contemporaneous accretional deformity, sixteen teeth at the most may be injured, the total of eight incisors and four cuspids, above and below, and the four first molars. The usual number damaged is less, whereas in a mottled enamel case, the entire dentures of vast populations, may be injured.

In considering the etiology of mottled enamel, it is again pointed out that the outstanding feature is its occurrence in geographical areas whose limits are sharply defined, and in all individuals who have maintained a continuous residence in such areas during the period of enamel development.

Unless the salient features associated with the production of this peculiar lesion are comprehended, it might be assumed that

some defect in the nutrition of the mothers was responsible. Under such a supposition the main feature in the etiology would still be unchanged, for the reason that it would be necessary to demonstrate that the mothers in an endemic district were uniformly subject to precisely the same nutritional or pathological dyscrasia, which would bring about the identical lesion of the enamel in their offspring. Such an assumption is not borne out by the facts. In the first place the process of enamel calcification of any of the permanent teeth does not commence until about, and probably not until after birth. Furthermore, it is a uniform observation that children born in non-endemic territory, but coming into endemic territory at say one year of age, will exhibit mottled enamel on the incisors and first molars when they erupt. To go further, children coming into an endemic district from non-endemic territory, say at the age of five to seven, will exhibit normal enamel on the incisors and first molars, but the bicuspid and second molars will be mottled. At such ages all pre-natal nutritional influences of the mother will long since have ceased. To go a step further, children coming into endemic territory at the age of say thirteen to fourteen, will exhibit mottled enamel on the third molars when erupted, the rest of the denture being normal.

Considering this question of the mother's influence conversely, a child born in an endemic district, but removed at say one year of age, will escape this damage to the enamel even though the mother or both parents might have been natives of the district and themselves afflicted with mottled enamel. Such evidence as this effectually disposes of the question of parental influence as related to the etiology of this lesion.

From these facts there is no other conclusion than that the injury is wrought by some influence contributed by an endemic district. Since the factor is so uniformly dystrophic in its influence, not associated in any known way with a pathology of other tissues, it is evident that it is contributed by some article of the nutritional supply that is locally produced, and the one article that most completely conforms to this specification is water. It might be argued that such other locally produced food as dairy products and vegetables should not be overlooked as a possible influence, but the evidence is so overwhelming as to rule out such a supposition. Certain communities can be pointed out, so barren in agricultural aspects as to make impossible the production of dairy or any other food products, in which 100 per cent of the native population had mottled enamel. The entire food supply of such communities is imported from other places in which mottled enamel does not exist.

In the attempt to locate a factor that was common to the water supply of endemic communities and localized districts, analyses of some of these waters were obtained, expressed in the usual form,

and without attempts to demonstrate the presence of the rarer elements. A table of comparative analyses made in this form, from three known endemic districts is given below.

TABLE No. 1
ANALYSES OF WATER FROM THREE ENDEMIC DISTRICTS.

Constituents given in parts per million	1 Water from a coal mine in Colorado	2 Mountain water Colorado	3 Water from a warm spring in Idaho
Sodium.....	276.77	3.30
Potassium.....	84.00
Calcium.....	89.75	5.70	2.70
Magnesium.....	27.36	1.70	.90
Iron.....49	.06
Aluminum.....	2.74
Chlorine.....	47.37	7.29	52.00
Sulphuric Acid.....	651.49	7.10	26.00
Carbonic Acid.....	216.78	15.30	19.00
Silicic Acid.....	16.68	7.50	78.00
Bicarbonate radicle.....	69.00
Nitrate radicle.....23

These waters, derived from varying sources, show marked differences in their chemical constituents, and yet each is associated with the production of mottled enamel. A theory that prevailed for a long time was to the effect that the failure of the enamel calcification was due to a lack of calcium in water. Reference to Column No. 1, in the above table, reveals a far greater calcium content than in No. 2, yet the injury to the enamel prevailing in the first district is uniformly more severe than in the second.

To further compare the calcium ratio in its relation to mottled enamel the table following is appended.

TABLE No. 2
ANALYSIS OF CUMMINGS' RANCH WATER, (WELL)

<i>Ions</i>	<i>Parts per million</i>
Sodium (Na).....	4.92
Potassium (K).....	2.67
Calcium (Ca).....	10.50
Magnesium (Mg).....	2.34
Iron (Fe).....	1.04
Aluminum (Al).....	2.32
Chlorine (Cl).....	2.40
Sulfuric acid ion (SO ₄).....	4.85
Carbonic acid ion (CO ₃).....	52.86
Silicic acid ion (SiO ₃).....
Organic matter.....
Total solids.....	83.90

Not only had several children born on the ranch used the water from this shallow well, but the mother also, since the age of two years. The enamel of all these persons was normal. The water

shown in Column No. 1, in Table No. 1 of comparative analyses (from coal mine) contains nine times as much calcium as that from the Cummings' well Table No. 2 and was associated with a 100% affliction of mottled enamel in that community, some cases being of unusual severity. Three of these are illustrated in Figures 36, 37, 38.



FIG. 36.

FIG. 37.

FIG. 38.

FIGS. 36, 37, 38. Colorado. Three brothers raised on water from coal mine. See Column No. 1, Table No. 1.

The amount of calcium contributed by water for use in bone or enamel formation is probably so small in amount, if any, as to warrant no consideration.

Table No. 3 contrasts the water from an endemic district with that from a district in which mottled enamel does not exist, the two communities being not more than four miles apart.

TABLE NO. 3

WATER ANALYSES CONTRASTING WATER FROM AN ENDEMIC DISTRICT WITH WATER FROM A NON-ENDEMIC DISTRICT.

Constituents given in parts per million	No. 1 Artesian water, Ark. <i>Endemic</i>	No. 2 River water, Ark. <i>Non-endemic</i>
Total residue on evaporation.....	1,003.00	86.00
Loss in ignition.....	43.00	14.00
Fixed residue.....	960.00	72.00
Chloride.....	415.90	3.75
Sulfate (SO ₄).....	36.60	15.70
Nitrogen as nitrate (NO ₃).....	.03	.03
Silica (SiO ₂).....	18.60	6.00
Iron and Aluminum oxides.....	1.00	.03
Calcium.....	25.30	17.06
Magnesium (Mg.).....	7.00	2.01
Sodium (Na).....	344.06	9.06
Potassium (K).....	9.02	3.04
Alkalinity (Phenolphthalain).....	1.00
Alkalinity (Methyl orange).....	213.07	52.00

Chemical interpretation of such analyses had never called attention to any one ingredient that could be assumed to be respon-

sible. There had long been a strong suspicion that these waters might contain some chemical property that could be revealed only by more refined methods of analysis.

In consideration of this lesion by others, it has been set forth that the damage was in the nature of a traumatic injury brought about by contact of the water with the teeth as in the act of drinking, during which calcium was "leached out" of the enamel. The repeated experience of observing erupting teeth with the enamel already mottled is sufficient to set aside this theory. The most



FIG. 39.



FIG. 40.

FIG. 39. South Dakota. Older sister, raised on shallow well water. Enamel normal.

FIG. 40. South Dakota. Younger sister, raised on deep well water. Enamel mottled.



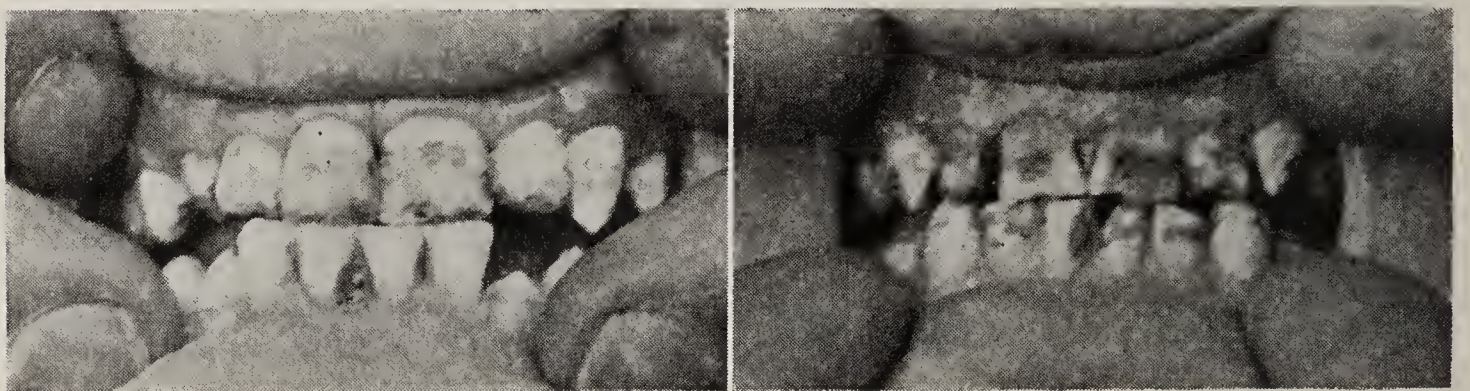
FIG. 41. Normal enamel in a child raised on shallow well water in an endemic district supplied by deep well water.

convincing proof is furnished by several third molars which are in the writer's possession that were completely impacted and when extracted found to be typically mottled. The enamel on these teeth was calcified in a known endemic district, from which the individual had been several years removed when the teeth were extracted, hence had never been touched by the "endemic" water. Such evidence supports the fact that this injury is exclusively developmental in its origin.

In setting forth the etiology, interest will be added by citing a few of the many specific examples that have been observed in

endemic districts, which will illustrate how definitely the water has been associated with this lesion.

The city of B, South Dakota, installed a municipal water supply from deep (drilled) wells which had been sunk about 1898, primarily to afford pressure for fire protection. Prior to that time each family had depended on its own shallow (dug) well. Following this deep well water installation, mottled enamel made its appearance. The two illustrations, Figures 39 and 40, show the teeth of sisters, natives of this city. Figure 39, the elder sister was born prior to the installation of the deep well, and therefore, was using shallow well water during the period of enamel calcification. The enamel is normal. Figure 40, the younger sister was born after the deep wells came into use and the teeth are typically mottled. Figure 41, shows the teeth of a child about eleven, the enamel on whose permanent incisors is perfectly normal. This child is one of five, all born on the homestead in this same city, and all having normal enamel. It seems that because of their dislike for the taste of the artesian water, it was not piped into the house,



FIGS. 42 and 43. Twins raised on deep well water. Enamel mottled.

the family preferring to continue the use of the 52 foot well in the yard, which had been in constant use for 28 years when our examination was made. Another interesting bit of evidence relating to the influence of this same well is furnished by the A family, who lived on the property next adjoining the homestead just described. Two children were raised there with constant use of water from this well, and have grown normal enamel.

Evidence more conclusive is afforded by the H family at K, South Dakota. Figures 42 and 43 show the teeth of twin boys born on the homestead the same year that a deep well was sunk to replace the shallow well previously in use for many years for the household supply. These boys therefore were raised exclusively on the deep well water, developing a mottling of the enamel of the severe corrosive type. Figures 44, 45 and 46, show the teeth of three older sisters in the same family who were respectively, fourteen, twelve and eleven years old when the deep well was sunk. The enamel of all the teeth, therefore, except the third molars, was grown during the use of the shallow well water, hence is normal.

The third molars however, of each of these sisters, were calcified after the deep well water came into use and were typically mottled. The four third molars of the youngest sister, Figure 46, had been extracted and are illustrated in Figure 47.

The endemic localities examined during the investigation have been with few exceptions small communities, surrounded by agricultural outlying districts. The children who live within the community and use the municipal water supply will invariably present



FIG. 44.

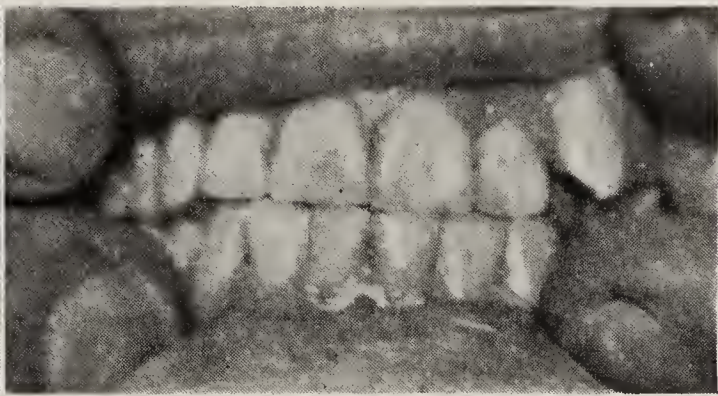


FIG. 45.

FIG. 44. South Dakota. Fourteen years old when deep well was sunk. Enamel normal except third molars.

FIG. 45. South Dakota. Twelve years old when deep well was sunk. Enamel normal except third molars.



FIG. 46.



FIG. 47.

FIG. 46. South Dakota. Eleven years old when deep well was sunk. Enamel normal except third molars.

FIG. 47. South Dakota. Third molars of Fig. 46, calcified during use of deep well water. Enamel mottled.

mottled enamel, and it has been a constant experience when examining children who presented normal enamel in such communities, to find that they resided outside the town and used water from other sources, their use of the municipal supply being limited to what little they drank during school hours. Apparently this dosage was too small to be of any effect on the teeth. These circumstances have been observed time and time again in the examination of many endemic communities. It cannot be seriously contended that the children within such communities have been uniformly subject

to the same pathological condition, or the same dietary errors, that might account for this lesion, while those outside the town were just as uniformly free from these. No more could disease or malnutrition account for a 100% incidence of mottled enamel in a given community, while a neighboring community, not over four or five miles distant be completely immune. The unvarying testimony in these endemic towns is that mottled enamel was unknown before the advent of the then existing municipal water supply, and in no one of these places has a native case been observed that antedated that supply.

FLUORINE IN RELATION TO THE PRODUCTION OF MOTTLED ENAMEL. The occurrence of fluorine, an element known to exert a destructive or disturbing influence on calcified animal tissues, as a constant constituent of waters associated with mottled enamel, was first established by H. V. Churchill, through a series of spectroscopic examinations of water from endemic districts.* It is only through years spent in studying the conditions in endemic districts, and the baffling attempts to locate in the waters an ingredient common to them all, that was capable of acting in this destructive way on the developing enamel, that the value of Churchill's work becomes apparent. Through it, this investigation was provided for the first time, with a definite aspect from which further studies of this lesion in its association with water, could proceed.

Churchill's determinations established, as far as they went, that no water containing less than two parts per million of fluoride was associated with mottled enamel, and further, that fluorides were absent, or present only up to one part per million, in waters from non-endemic districts. The highest fluoride content in any water examined up to the time of writing is sixteen parts per million. It seems also to have been established that the higher the fluoride content the more severe is the injury to the teeth, particularly as regards the corroded phase of the lesion. The physical character of these waters conveys no indication of their disturbing effect upon enamel calcification, being free of sediment, generally agreeable to the taste, odorless, and satisfactory from all other standpoints for drinking, cooking and general domestic purposes. Fluorides and other minerals are apparently in complete solution.

The literature of biological research records various experiments with laboratory animals through which fluorides introduced into the ration have been found to be injurious to the calcified tissues including the teeth. The reports of these experiments, however, have not been specific as to the exact character of the injury

* Churchill, H. V. "Occurrence of Fluorides in Some Waters of the United States." Synopsis in the News Edition, *Journal of Industrial and Engineering Chemistry*, April 10, 1931, Vol. 9, No. 7, p. 105. *Journal of Dental Research*, 1932, Vol. 12, p. 141.

done to the dental tissues, but it has not been inferred that the lesions produced in that way were similar to, or bore any relation to mottled enamel.

The fluorine content of the water in its relation to mottled enamel has received biological confirmation through a remarkably thorough and conclusive study conducted by Smith and her co-workers at the University of Arizona.* During a series of experiments, concentrates from the evaporation of water from an endemic district in Arizona that was found to contain fluorine, were fed to laboratory animals, resulting in a production of a lesion stated to be essentially identical with mottled enamel. These experiments were further complemented by feeding rations to laboratory animals into which fluoride had been incorporated, without relation to any particular water, with the same results on the enamel. Smith and her associates have carried their studies further by making a complete survey of every sizeable community within the State of Arizona. In every community in which mottled enamel occurs the water was found to contain upward of two parts per million of fluorine, and in those communities in which the fluorine was below that proportion, mottled enamel did not exist.†

Fluorine as a constituent of water is a problem that has heretofore received scant attention from water chemists and water works engineers, consequently there seems to be little known concerning it. It can only be assumed, therefore, that its action is mildly toxic, at least in the dosage administered by the water in drinking and cooking, but sufficiently so to interfere with the process of enamel calcification and to damage the cells of that tissue. Concerning the enamel, it would seem that fluorine exhibits an amazing specificity provided it can later be demonstrated that no other cells or tissues are injured. So far as is known at the present time there is no damage to other tissues in residents of districts in which mottled enamel is endemic.

The evidence then indicates that the presence of fluorine in the water in proportions of approximately two parts per million and above, is the one factor that accounts for the production of mottled enamel. Communities and districts in which this situation exists are faced with the problem of obtaining water from another source, that shall be free of fluorine, or of undertaking a chemical modification of the existing supply that will eliminate or nullify the action of fluorine if the further production of mottled enamel is to be brought under control.

* Smith, Margaret Cammack; Lantz, Edith M., and Smith, H. V. "The Cause of Mottled Enamel, A Defect of Human Teeth." University of Arizona, Technical Bulletin No. 32, June 10, 1931.

† Smith, Margaret Cammack, and Smith, H. V. "Mottled Enamel in Arizona and its correlation with the concentration of Fluorides in Water Supplies. Technical Bulletin No. 43, University of Arizona, July 15, 1932.

At the moment there seems to be no method available for accomplishing this latter on a scale large enough to be applicable to a municipal water supply. Individual families might resort to distillation, or might import safe water for household use as a means of protection to their children.

GEOGRAPHICAL DISTRIBUTION.

The investigation of this lesion has disclosed that it occurs in widely scattered districts of the United States, and in certain other parts of the world, and in association with water supplies drawn from almost every conceivable source, the most constant relation, however, being with water from deep or artesian wells.

It is not to be inferred that all artesian or deep well waters are necessarily productive of mottled enamel, but only that they are to be looked upon with suspicion, particularly in regions in which this lesion prevails. Apparently mineral deposits bearing fluorides are not often encountered in surface outcrop adjacent to the sources of municipal water supplies, although one instance is known where this has happened. These deposits are usually at deeper levels through which the waters from artesian wells and warm springs percolate.

Throughout the United States, districts endemic to mottled enamel are known to exist in Arizona, Arkansas, California, Colorado, Idaho, Illinois, Minnesota, New Mexico, North and South Dakota, Iowa, Texas, Virginia, Kansas, and reliably reported also in Nevada, North Carolina, Oregon and Washington. It will be noted that with the exception of Virginia and North Carolina, none of these States lie east of the Appalachian range, and confirmatory evidence bearing on the fluorine relation is given in Churchill's findings, that no municipal water East of the Appalachians, examined by him contained more than one part per million of fluorine.

By far the greater number of the afflicted districts in the States named, derive the water from deep wells, although in certain places water from warm springs has been used, and in two known localities water drawn from coal mines. It must be admitted that water from a warm spring or from a coal mine is an unusual source for a community water supply, but in both these instances such water had served the purpose adequately except for its interference with enamel calcification.

Outside the territorial boundaries of the United States there are endemic districts in the Bahama Islands, Barbados Island, the Island of Brava in the Cape Verde group, China, The Netherlands, Italy, Mexico, North and South Africa, South America, particularly Argentine, Spain and England. There is very little definite knowledge available at the time of writing concerning the character of the water supplies of any of these districts.

The occurrence of mottled enamel in the Bahamas is apparently limited to certain of the Islands, particularly, Rum Cay, Watling's and Ragged. The water is drawn from shallow wells sunk into the coral of which these islands are chiefly composed.*

The water that has been associated with the production of mottled enamel in The Netherlands, is, according to the best testimony available, derived from the sand dunes.

The district examined in Italy is adjacent to the city of Resina, which overlies the lava flow erupted from Vesuvius in A. D. 79, which buried the ancient city of Herculaneum, and the mottled enamel cases found, had used water from wells of a depth of about 50 feet, that penetrated this underlying lava flow.†

There is little doubt that this lesion is distributed among inhabitants of the Earth to an extent that is at the present time not fully realized. It is not to be inferred that sporadic cases occurring in individual persons may not be found almost anywhere, but in the writer's experience they are extremely rare and difficult to account for. G. V. Black had noted a few such instances in the previous editions of "Operative Dentistry". Finding such an isolated case now and then is entirely different from an endemic occurrence, involving entire communities.

TREATMENT.

To practitioners not in or adjacent to districts endemic to mottled enamel, this lesion is of little if any clinical interest, but to those in daily contact with it, any method of treatment that will remove or modify the stigma of its appearance is of practical and absorbing interest.

In undertaking measures for the restoration of tooth tissue lost through caries, the establishment of cavity margins in these teeth, is difficult because of the chalky texture of the enamel. In extreme cases of the corroded phase of the lesion the most practical and esthetic restoration would be the porcelain jacket crown, but practically any case of moderate intensity, either as to the aspect of corrosion or stain, and even cases of pronounced intensity, can be treated with most gratifying results in improved appearance by the method about to be described.

In Black's description of the histology of this lesion and the illustrations, Figures 34 and 35, it is seen that the defective and stained enamel occupies the outer one-fourth to one-third of the

* McKay, Frederick S. "Progress of the year in the investigation of mottled enamel, with special reference to its association with artesian water." *Journal of the National Dental Association*, 1819, Vol. 5, p. 721.

† McKay, Frederick S. "Mottled enamel; a report of an examination of an afflicted district in Italy." *Journal of Dental Research*, 1928, Vol. 8, p. 353. Abstract: *Ibid.*, p. 433.

tooth surface, and that underlying this the structure became increasingly more normal approaching pulpward. The treatment then would be the obliteration of this outer layer, finally coming down to what is practically normal enamel. This can be accomplished in two ways, either by chemical dissolution, using mineral acids, or mechanically, by means of grinding.

From time to time certain processes have been proposed which made use of dissolving fluids, but as these processes have been kept

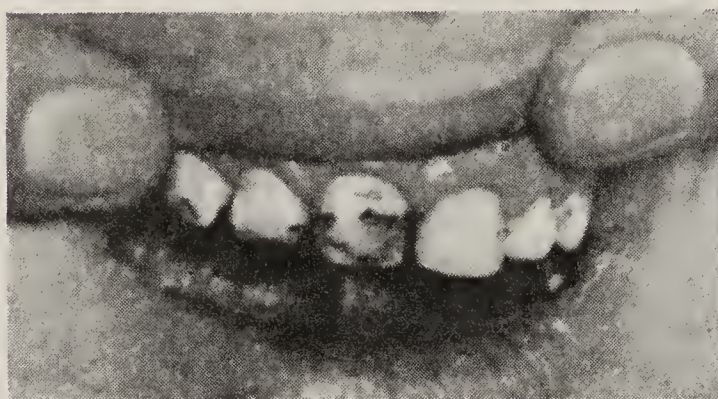


FIG. 48.

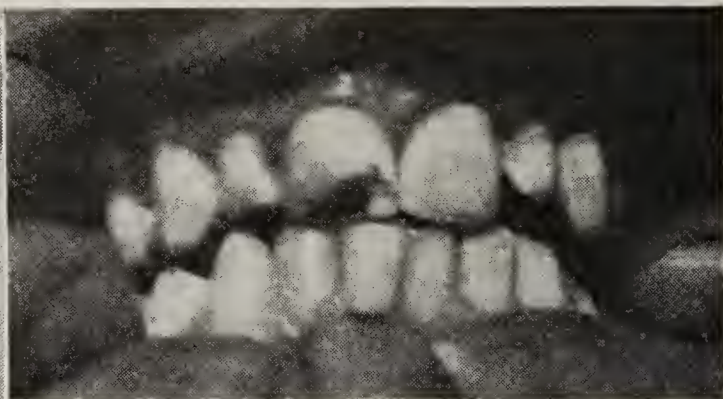


FIG. 49.

FIG. 48. Right incisors before treatment; left incisors after treatment. These were similar to right incisors before being treated.

FIG. 49. Upper right central incisor before treatment, left central incisor after treatment: this was similar to right central before being treated.



FIG. 50.



FIG. 51.

FIG. 50. Upper incisors before treatment.

FIG. 51. Same case after treatment of upper incisors.

secret, it can only be assumed that such fluids were mineral acids. Knowing no more than this concerning them, their description cannot be given here. Leaving aside the danger of using a mineral acid on such porous enamel tissue, it makes little difference in a practical way, as the treatment requires the destruction of the outer defective layer, whether this be accomplished chemically or mechanically.

For the reason that the treatment of a given tooth or group of teeth can be completed in one sitting and does not involve the

dangerous and questionable use of an acid, the writer prefers the mechanical method by grinding. See Figures 48, 49, 50, 51, 52 and 53.

The labial surface of a tooth to be treated is dressed down uniformly by means of a fairly coarse stone, preferably of a barrel or conical shape, kept well moistened with water, applying the stone in a sweeping motion from mesial to distal, and incisogingivally, until the greater part of the defective tissue has been removed. The sweeping motion of the stone over the entire surface is to avoid leaving depressions. The tooth surface should be dried at intervals with compressed air, and examined with a magnifying glass. This will indicate the progress made.

This process should be continued until the underlying layer of normal enamel has been reached, except that here and there a few flecks of white enamel may remain, which will not be visible when



FIG. 52.

FIG. 52. Before treatment.

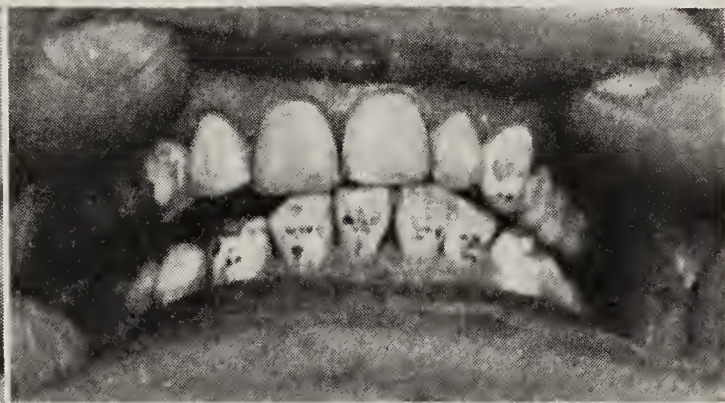


FIG. 53.

FIG. 53. Showing upper incisors of same case after treatment. Later the entire incisor and cuspid groups of teeth above and below were treated with similar results.

the tooth is wet. The complete removal of these might involve too great a loss of normal enamel, as these white flecks indicate spots where the defective enamel penetrates or dips more deeply into the substance of the normal enamel. At this stage, the stain should have been completely obliterated even in pronounced cases.

The most difficult cases are those in which the stain occurs as a sharply defined band across the tooth surface with the enamel comparatively normal both incisally and gingivally. The treatment of such a case may involve leaving a shallow depression at the location of the stain, but rather than encroach too closely upon the dentin it may be necessary to leave a faint trace of the discoloration. Judgement must be used in any instance, but experience has shown that there is no case that cannot be astonishingly improved by this treatment.

The first coarse stone is followed in the same way by finer ones to remove the scratches, and these in turn by sand paper discs of medium grit, put in the engine mandrel double, back to back,

and well lubricated with vaseline to avoid heating, as the enamel becomes appreciably thinned. Finer discs are used to obtain a high polish. The proximal surfaces should be dressed down with abrasive strips, starting with fairly coarse ones and following with finer grits, finishing with a silk tape charged with wet pumice and then with chalk.

After the disking has been completed, a buff wheel of leather or felt with wet flour of pumice should be used, followed with wet prepared chalk on a different buff wheel; the final high lustre can be produced with dry chalk on the same buff wheel.

All surfaces of all teeth can be treated in this way, and all pits and defective cusp points can be smoothed out and polished. The experience upon which this method is based covers literally scores of teeth and no damage to any tooth so treated has been observed. In some cases there may be a sensitiveness to changes of temperature for a time which eventually disappears.

To demonstrate the results of this method a series of illustrations is introduced.

THE PREVENTION OF FURTHER PRODUCTION OF MOTTLED ENAMEL BY A CHANGE IN THE WATER SUPPLY AT OAKLEY, IDAHO.

Up to the time when this is being written, two communities in the United States, Oakley, Idaho, which had used water from a warm spring, and Bauxite, Arkansas, which had used water from artesian wells, have discarded these supplies and substituted others from different sources solely on account of this enamel defect.

Following an examination of the public school children of Oakley, conducted by the writer on February 9th, 1925, which revealed that mottled enamel existed in every child who had used the warm spring water through the period of enamel calcification, that supply was abandoned and water from a known non-endemic source was installed for use about the first of July of the same year, five months later.* On February 6th, 1933, eight years after the original examination, and approximately seven and a half years after the change in the water supply, a re-examination of the school children was made in which the writer was assisted by Dr. H. B. Smith, of Jerome, Idaho, who assisted also at the original examination.

The children were classified into groups according to the ages that had been attained at the time of the change of water. In the youngest group, born one and a half years after the change there were eight; in the group born six months after the change there were two; in the group born at about the time of the change or just before, there were fifteen.

* McKay, Frederick S. 1925. "Mottled Enamel: a Fundamental Problem in Dentistry." *Dental Cosmos*, 1925, Vol. 67, p. 847.

It is obvious that the twenty-five children in these three groups were raised exclusively on the new water and it was found that all of the permanent teeth that had erupted, 161 teeth in all, showed no trace of the defect. The ages in this group, at the time of the examination, in no cases exceeded just under eight years. Figures 54, 55 and 56 illustrate the teeth of three children showing permanent incisors on which the enamel is normally calcified, with the exception of Figure 54.



FIG. 54.



FIG. 55.

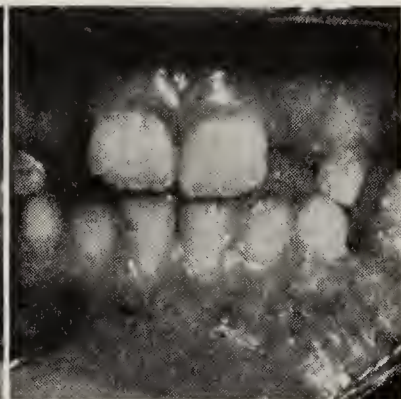


FIG. 56.

FIG. 54. Oakley, Idaho. Child was eight months old when the water supply was changed. First permanent molars mottled slightly on cusps and upper central incisors slightly on incisal portion.

FIG. 55. Oakley, Idaho. Child was six months old when water supply was changed. Enamel on all erupted permanent teeth is normal.

FIG. 56. Oakley, Idaho. Child was born six months after water supply was changed. Enamel on all erupted permanent teeth is normal.



FIG. 57.



FIG. 58.

FIG. 57. Oakley, Idaho. This child was 4½ years of age when the water was changed. Note the line of demarcation on lower incisors.

FIG. 58. Oakley, Idaho. This child was 5½ years of age when the water was changed. The mottling is generally distributed over all teeth, but mild in degree. Two small stained areas are seen on the upper central incisors.

These illustrations can be contrasted with Figures 15E, F, G and 19, which illustrate the damage prevailing among Oakley children raised on the former warm spring water as determined at the original examination in 1925. In a group of eleven children who had attained the age of six months at the time of the change, two showed a very slight mottling of some of the incisors and first molars, all other erupted teeth in the group being normal.

In passing through the groups that had attained ages of from one year up to two and a half years at the time of the change, there was found to be a generally prevailing normality of enamel structure, with a few exceptions in certain incisors and first molars which were very slightly mottled, but in coming to the groups that had attained the age of two and a half years and upward at the time of the change, there is an increasing prevalence of the defect with a general involvement of more teeth.



FIG. 59.



FIG. 60.

FIG. 59. Oakley, Idaho. This child was 5½ years of age when the water was changed. Note line of demarcation on lower bicuspid.

FIG. 60. Oakley, Idaho. This child was 6½ years of age when the water was changed.

In these children the effect of the use of the former warm spring water was clearly apparent, but even in the oldest of these overlapping cases, the damage inflicted was in no way comparable in its severity with that prevailing under the exclusive use of the former water supply.

Figures 57, 58, 59 and 60 show the most extensive damage that could be observed among those children in whom the period of enamel calcification had overlapped the change.

The examination included 99 children who presented a total of 954 normal teeth. The evidence was conclusive that since the adoption of the new water supply the production of mottled enamel had definitely and completely ceased.

The fluorine content of the former water supply was 6.0 parts per million, while the content of the new supply has been determined to be not above one-half of one part per million, which fact, together with the conditions found in the examination, affords a final conclusiveness as to the causative relationship between fluorine and the occurrence of mottled enamel.

THE RELATION OF MOTTLED ENAMEL TO CARIES.

It is worthy of note that at the time of Dr. Black's first personal contact with this lesion in an endemic district, it had not been many years since the controversy concerning the relation

which the quality of the enamel structure bore to the liability to dental caries, was at its height.

That was an historic episode in the progress of dental science and was marked by strong differences of opinion on both sides of the question. The prevailing opinion during that era was that the liability to caries was largely controlled and dependent upon the integrity of the enamel structure in reference to its degree of calcification; well calcified or completely calcified teeth were considered as less liable to decay than were "poorly" or "imperfectly calcified" teeth.

Black's dictum promulgated at that time, that there was very little if any difference in the degree of calcification of enamel, as indicated by its specific gravity, hence that factor could not be accounted as the chief influence in the etiology of caries, aroused a storm of dissenting opinion. It was only natural, therefore, that at the time of his personal examination of a district endemic to mottled enamel, he should be impressed by the fact, which he recorded, that the teeth exhibiting this defect seemed not to be more subject to carious involvement than teeth on which the enamel was normal.

The investigation had proceeded on its way several years before it was recognized that this fact contributed in an important way to the etiology of dental caries, because of the wide previous acceptance of the belief that the degree of calcification was the chief factor influencing the liability to decay. Mottled enamel is the most poorly or incompletely calcified enamel of which there is record in the literature of dentistry, therefore, according to the prevailing idea just referred to, its liability to decay would be considered high.

In order to reach a determination on this point the more recent examinations of endemic districts have included a careful record of carious involvements of the teeth in addition to the usual observations connected with mottled enamel. In these examinations the chief effort has been to determine if those teeth with mottled enamel were, because of that defect, thereby more liable to decay than teeth with normal enamel in the same community. Up to the time of writing, the public school children in four communities, one in Idaho; one in Colorado; one in Arizona;* and one in Kansas, have been examined in this way. In the three first named there was actually found to be less decay in the mottled teeth than in the normal, and in the last one named the percentages were about equal.

* McKay, Frederick S. "The Establishment of a Definite Relation Between Enamel that is Defective in Its Structure, as Mottled Enamel, and the Liability to Decay." *Dental Cosmos*, 1929, Vol. 71, p. 74; also *Pacific Dental Gazette*, Vol. 37, p. 599.

No comment on these results is to be made except the main fact that in all these places mottled enamel was not found more liable to decay than normal enamel.

In many individuals examined both mottled and normal enamel were found in the same mouth, subject to the same environment, with no difference in the incidence of decay. Conforming to common experience, in these examinations by far the greater number of decays were found to be in the pits and fissures, and in this respect the mottled teeth presented precisely the same evidence as did the normal teeth. The defective, poorly calcified enamel on the mottled teeth did not render these teeth liable to decay on other than the well known vulnerable surfaces and areas on which normal teeth decay.

The above then would seem reasonable evidence that the quality or degree of calcification of the enamel cannot be offered as an etiological factor in determining the incidence of decay. This consideration of mottled enamel seems to have established the following points that are worthy of emphasis in recapitulation.

Structural defects in enamel can be inflicted only during the formative or calcification period, and when once inflicted persist in a static condition throughout life. No repair processes are apparent through natural means and no further deterioration of the same nature takes place.

Even at the height of its developmental activity during the calcification process, the enamel seems incapable of overcoming the toxic or disturbing effects of a nutritional error such as that which produces mottled enamel.

Enamel after eruption seems not subject to damage by the same influence that inflicts injury during its developmental process.

There is no other known specific lesion of the enamel, uniformly presenting such definite histological characteristics, that is so directly associated with a dietary error as is mottled enamel.

Considering the disturbing constituent of the water as a dietary error, it invariably produces the same lesion of the enamel in all individuals who use the associated water during the period of enamel development, and in whatever part of the world it occurs, regardless of all other dietary influences.

HYPOPLASIA OF THE TEETH

ATROPHY OF THE TEETH

ILLUSTRATIONS: 68-109.

HYPOPLASIA, or atrophy of the teeth consists of a failure of the formation or an imperfect formation of some specific portion of the tooth and of several teeth together. The portion of the several teeth affected is always that portion of each that was in process of formation or growth at the same period of the person's existence.

This deformity of the teeth is always caused by illness that has interfered with nutrition at the time the particular parts of the teeth affected were in process of calcification.

Hypoplasia is a contemporaneous accretional deformity — a dystrophy in which all portions of the teeth in process of formation at a particular time are imperfectly formed along the lines of normal accretion or growth.

In the previous editions of this work, the term *atrophy* was applied to this condition, also the term *hypoplasia*, which has been used widely in the German writings. Because of the fact that the application of both of these terms has been too general, has included all kinds of deformities, and also because of the fact that the term atrophy represents two distinct ideas in medical literature, it seems desirable that both terms should be dropped as applied to this condition. The term atrophy has been applied to a failure of development of a local part because of a failure of nutrition; it has also been applied to the wasting of a part because of a local failure of the nutritive process. The term atrophy has been used to designate this condition since it was first spoken of in the English language.

The contemporaneous accretional deformities of the teeth represent an atrophy of the first mentioned type; they have never been fully formed. They come through the gums in the condition of deformity in which they are afterwards seen, and do not waste away after having taken their place in the arch. The deformity is a result of incomplete formation, and the trend of the present day appears to favor the term hypoplasia, which means under development.

In the human teeth there is no process of repair and the deformity is permanent. Similar phenomena occur in the finger nails and the hair. During an illness that interferes seriously with nutrition, the portion of the finger nail then forming will be dwarfed, which will appear later as a groove across the nail. This, like the marking of the teeth, is not remedied by any reparative process. But the nail is continuously growing and the groove moves on over the length of the nail and disappears.

During a severe illness that interferes with nutrition, a section of hair is imperfectly formed, and when in the process of growth this section arrives at the surface of the skin and is subjected to bending, it breaks and the hair suddenly falls away. These are common phenomena following severe cases of typhoid fever. The hair follicles are not injured and the hair is replaced by the regular process of growth.

In the continuously growing teeth of the rodents such an injury would be finally removed and remedied in the same way as the grooving seen upon the finger nails, but this can not occur in the human teeth.

The deformity, though much varied in different cases, is, when closely analyzed, always similar in character. It always consists in a failure of the formation or an imperfect formation of specific portions of several teeth that were in process of formation at the same time. To understand this well one should study closely the calcification of the crowns of the teeth and the contemporaneous lines of calcification of the different teeth. This will be more fully explained in considering the histological changes occurring in hypoplasia. In the incisors the deformity is oftenest seen in the form of a groove, smooth or pitted, running across the labial surface from mesial to distal, and close inspection will generally show that it encircles the tooth completely, though it is most prominent upon the labial surfaces where the enamel is thickest. It is seen more often on the incisal half of the length of the crown. It may be near the cutting edge of the tooth or anywhere from that point toward the cemental line. It is also found occasionally in the roots of extracted teeth. There may be a single groove or pitted line, or there may be two or even three or more of these. The teeth affected are the incisors, cuspids and first molars of the permanent set, and very rarely the first bicuspid. It rarely, if ever, affects the temporary teeth. If it is very close to the cutting edge on the central incisors it may not appear on the laterals, but the occlusal surfaces of the first molars will be affected. This is because these parts of these teeth are in process of formation at the same time. If the groove is a little further removed from the incisal edge of the centrals, the lateral incisors will also be similarly affected. If it is a little higher still, the four incisors, upper and lower, the cuspids

and the first molars will be affected, but the bicuspid will be free from injury. It is exceedingly rare that the bicuspid or the second or third molars are affected, for the reason that the enamel and dentin of these teeth generally have not begun to form until after the age at which these effects are most liable to occur. But few cases occur in which the first bicuspid is marked. The time of the occurrence of these injuries seems to be confined mostly to the first five years of a child's life, but some cases occur later.

In the engravings the endeavor has been to illustrate some of the more severe types of these deformities and to explain by illustration the histological defects. Figure 68 represents what is known as the typical Hutchinson tooth, from the claims of Mr. Hutchinson, a specialist in venereal diseases in London, England, who at one time believed that this deformity was the result of congenital syphilis. In such cases it has formerly been supposed that the middle lobe has failed of formation, resulting in this peculiar scar, but more recent investigation seems to show that the whole incisal edge has failed in most of these cases, and that the angles of the tooth have been drawn together over the injury, giving the outline of the tooth this rounded appearance. Certainly

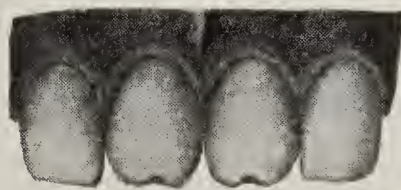


FIG. 68.

FIG. 68. Hypoplasia of the cutting edge of the central incisors, forming a central notch or scar. This is usually called "Hutchinson's tooth." In connection with this form of hypoplasia, the occlusal surfaces of the first molars are also badly marked.

many of these teeth are much shorter than normal. Generally a severe deformity of the occlusal surfaces of the first molars accompanies this type. In the molars little spiculæ of cusps are likely to be sticking up much too close together, while the rest of the occlusal surfaces are much too small, crumpled together, and sunken into the crown, which, other than this, will be of full size and form. These teeth decay quickly in case there is a tendency to caries in the individual.

Of the incisors shown in this illustration, only the centrals are affected. The calcification of the cutting edges of these is occasionally just begun at birth, and if not begun then, is usually begun within one year. The injury, therefore, occurs soon after the birth of the child from some cause which interferes with nutrition. A very curious fact in pathology is rendered prominent in this form of defect. It is this: when the nutrition of any single part of the enamel organ is so impaired that its function is stopped or very

seriously disturbed, that particular part does not recover, and no additional enamel is formed by that part. This will appear more prominently in the histological specimens. It is for this reason that these teeth have the peculiar rounded appearance of the cutting edge. The enamel organ of that part is arrested at the very beginning of the calcification, and therefore the immediate incisal edge fails entirely. The rest of the organ goes on with its work after the recovery and the tooth is drawn in over the scar. In the incisors this form of defect is apt to be attacked by decay in this incisal pit very soon after the teeth have taken their places in the arch. They should receive attention at once if decay is discovered. This particular form of hypoplasia is seen less frequently than others.

The occlusal surfaces of the first molars are occasionally badly deformed when the incisors have escaped. Usually these have just begun their calcification at birth, and occasionally the calcification of the central incisors does not begin for one year after birth. In such cases a severe illness may injure the molars and not injure the incisors.



FIG. 69.



FIG. 70.

FIG. 69. Hypoplasia of the cutting edge of the central and lateral incisors. When this occurs, some portions of the occlusal surfaces of the first molars will also be injured.

FIG. 70. Hypoplasia of the incisors and cuspids. In this case there is a pitted groove around the crown of each atrophied tooth. The first molars have a similar groove on their axial surfaces.

Much the more common forms are those illustrated in Figures 69 and 70, the illustrations showing rather bad cases. In Figure 69 the deformity is confined to the cutting edges, apparently, of the central and lateral incisors above and below, and the four first molars. In the case here illustrated the whole of the incisal edge of each of the incisors above and below is dwarfed and shortened. This dwarfed portion ends abruptly toward the gingival. This is common in these cases. In many there is more or less rounding down of the well-formed part of the crown to the deformed part, but often it is so abrupt as to form a square shoulder along which there is apt to be a series of sharp, deep pits. In the case from which Figure 69 is taken there are no pits whatever, and the deformity consists purely in the dwarfing of the incisal edges. But

the entire occlusal surfaces of the molars were in very bad condition because of dwarfing that presented many abrupt fissures in which decay began almost immediately after they had come through the gums. In such cases as this the appearance of the incisor teeth may be much improved by grinding away the dwarfed portion and shortening the cuspids a little to correspond with them. The teeth may appear a little short but that is sometimes much less noticeable than the blemish.

In other cases, occurring in the same locality and affecting the same teeth, there may be but little dwarfing of the incisal edges of the incisors. The effect may be but a slight groove that may be smooth or more or less pitted, or in cases of a still milder type the distinct groove may be lacking and a row of fine pits in the enamel will be the only deformity. Generally, the effect is more marked in the occlusal surfaces of the first molars than in the incisors.

In the case illustrated in Figure 70 the deformity occurred later, when the child was between three and four years old. The incisal portion of the incisors had been formed, and, therefore, there is no dwarfing of this portion of these teeth. But there is



FIG. 71. Hypoplasia of the incisors, showing the "inverted fingernail" scar on the lateral incisors.

a very marked groove encircling the crowns of the incisors and cuspids, marked with pits, with smoothly rounded bottoms. Both the groove and the pits in the groove are abrupt toward the gingival and thin away toward the incisal. This is a constant characteristic of these deformities, which will be readily understood by a study of the histological sections and the calcification lines of Retzius. The circular form of this deformity, as it passes from tooth to tooth across the front of the mouth, is well marked in Figure 70; indeed, it presents rather more of the circular form than usual, indicating especially that the cuspids were a little later than usual in their calcification, and for that reason the mark is nearer the incisal edge in proportion to the position on the incisors than it would otherwise have been.

So severe a mark as here shown is not frequently seen so high upon the labial surfaces of the incisor teeth. It seems to be a general rule that the higher upon the teeth the less marked is the deformity. Pretty generally, in this position on the centrals, the

mark is a shallow groove, more or less pitted, or a row of pits without a distinct groove. In all of these cases the lower teeth bear marks similar to those in the upper.

In Figure 71 a case is illustrated that is somewhat out of the usual form in several particulars. When the impression for the cast from which the illustration was made was taken, the cuspids had not come through the gums, but one of the first bicuspid had erupted, and showed a deep mark encircling the point of the buccal cusp. Also the history of the case shows that the lateral incisors did not erupt for two years after the centrals had taken their places. In the centrals the incisal edges are fully formed, but there is a deep groove with rounded pits encircling the crowns at nearly mid-length, while nearly the whole incisal half of the lat-



FIG. 72.



FIG. 73.



FIG. 74.

FIGS. 72, 73 and 74. Single teeth, the crowns of which have been badly dwarfed by hypoplasia. Each of these show two zones of injury. The dotted lines are intended to show the normal outlines of the crowns. Figures 72 and 73 are different views of the same tooth.

erals is badly deformed. This indicates that the beginning of the calcification of these teeth was late, as compared with that of the centrals. This particular form of deformity of the lateral incisors is not very frequent, but yet a considerable number have been seen, quite enough to indicate a tendency to this particular deformity. In the common vernacular this has been called the *inverted finger nail deformity*. If we imagine the finger nail taken up and turned with the convex side down and set back in the end of the finger, we would have something very like this deformity. The whole appearance of this case indicated unusual irregularity of the time of calcification and eruption of the different teeth. The first molars, both above and below, had already been destroyed by decay, beginning in the deformity of the occlusal surfaces.

Figures 72 and 73 show a lower incisor with a double deformity. Figure 72 is a view of the labial surface, and Figure 73 of the mesial surface. The dotted line shows the normal tooth form. The two, taken together, show the extent of the dwarfing of the crown of the tooth. In this case the surface of the enamel was smooth and without pits.

Figure 74 shows an upper central very badly deformed. This is also a double deformity and was further injured by decay starting in pits in the abrupt portion of the groove nearest the incisal. The sharp, deep pits shown along the line of the second groove



FIG. 75.



FIG. 76.

FIG. 75. Hypoplasia of incisors and cuspids showing no discoloration. Photographed direct from mouth of girl eighteen years of age.

FIG. 76. Plaster model showing a severe deformity of the incisors.

have not been caused by decay, but were there when the tooth came through the gums. These teeth are from different persons, and in both cases were extracted in order to remedy the defect with artificial teeth. This seems to have been done under the mistaken notion that the roots of the teeth would not be good for artificial crowns. Extended observation shows that the roots of such teeth are as apt to be well developed and as good for crowning as those of any other teeth. When the crowns are so badly deformed that it is out of the question to employ restorative procedures, porcelain jacket or other types of crowns should generally be resorted to, rather than to remove the teeth. In case the crowning involves the removal of the pulp, this should be delayed as long as possible in order that the roots may be fully formed and the apical foramen reduced to a small size. One should wait until the patient is sixteen to eighteen years old, and twenty would be still better. In many cases of considerable actual deformity of the teeth the color remains very perfect; if the incisal edges of the incisors are fairly complete, the deformity is not prominently noticeable, as is shown in Figure 75, from a photograph taken directly from the mouth.

Figure 76 is a reproduction of a photograph of a plaster cast which shows a very severe deformity of the incisors. The occlusal surfaces of the first molars were also poorly formed, although this is not well shown in the illustration. Figures 77 and 78 are two views of an upper central incisor, which is badly deformed.

ETIOLOGY.

This deformity is the result of malnutrition. It has been rather generally believed and so stated in many writings on venereal disease, that hypoplasia of the teeth is a sign of congenital syphilis. Hutchinson, of London, evidently thought so, but later



FIG. 77.

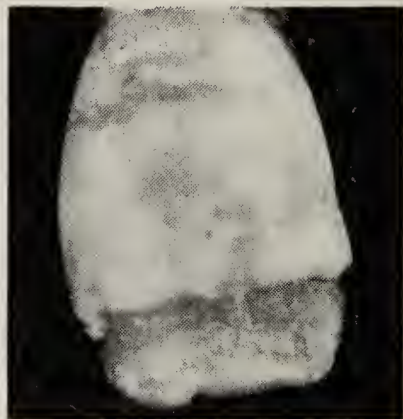


FIG. 78.

FIGS. 77 and 78. Two views of a badly deformed upper central incisor.

yielded the point so far as to say that congenital syphilis was a frequent cause, and for many years held that the type shown in Figure 71 was always caused by congenital syphilis. That form, therefore, has been called the Hutchinson tooth. As showing how errors are liable to be perpetuated, most of the books on general medicine, surgery and venereal diseases, which mention these deformities at all, ascribe them to congenital syphilis, following the first writings of Mr. Hutchinson without further investigation.

The author followed this subject pretty carefully from Hutchinson's time, adding observation after observation, and arrived at the conclusion that there is no particular form of disease that is especially responsible for this affliction, but that any form of disease that seriously interferes with nutrition is liable to bring about this result, i. e., that it is not the particular form of disease, *but that it is the condition of malnutrition that is the cause*, no matter what the disease which has induced that condition. Cases of typical Hutchinson teeth occur which are certainly in no way connected with a syphilitic taint of any kind.

Some of these observations may be of interest. Mr. and Mrs. B., known by the author intimately from childhood, had a child

which seemed healthy at birth, but soon afterward became anemic and did very badly for two years. Growth was a failure during that time, and it was with great difficulty that the child was kept alive. In its third year, however, the child recovered and became strong and healthy and developed well. When the permanent teeth came through the gums, almost the incisal third of the centrals and laterals was badly dwarfed, the points of the cuspids had failed and the occlusal surfaces of all of the first molars were badly deformed.

A child two and a half years old survived a severe case of typhoid fever. When the permanent teeth came through they were marked with a deep groove, irregularly pitted, similar to Figure 70, but not so high up on the crowns.

An English woman brought her child of seven with a bad deformity of the upper central incisors which had just come through the gums. In reply to inquiries she could not remember that the child had had an illness of any kind. The boy had always been healthy and had escaped all of the infantile diseases. Being convinced that something had occurred that would have been noted, inquiry was made as to a possible injury. This quickly brought out the statement that the child had had a severe burn, a scald, on the side and back, that had healed slowly after much suppuration. Indeed, the child had been very ill with septicemia for a month or six weeks. The time corresponded with the marks upon the teeth.

Cases like these, but every one different in detail, could be multiplied almost indefinitely. Scarlet fever, measles and whooping-cough come in for a large share in producing these marks, and certainly congenital syphilis causes many cases.

On the other hand it can not be said that hypoplastic teeth will result from this or that illness. If the hair falls out or the finger nails show a groove after an illness, the teeth may be malformed. Many cases in which there is definite history of severe illness previous to the fifth year, have presented teeth without a blemish. Indeed, deformity of the teeth is the exception rather than the rule. Still, it remains true that in every case presenting with this deformity, in which a history of illness could be obtained, the deformity has coincided in time with some form of disease that might well have interfered seriously with the nutritive processes. The history is not always easy to get, even among intelligent people. A woman, whose teeth showed definite deformity of this type, was told that she was ill when about two years of age, probably with scarlet fever. She was quite sure she had not been sick at that time. When it was explained to her that accurate knowledge of the facts was of considerable scientific value she said she would question her mother regarding it. The next day she re-

ported that her mother's story agreed both as to the particular disease and the date of the illness. Many of these cases, however, give no history of the illness causing them.

To assist dentists in searching for the cause that has led to this deformity in cases coming before them, a diagrammatic chart, or index, is shown in Figure 79. Lines across the incisors, cuspids and first molars, indicate the positions the grooves across the teeth assume because of disease occurring at different ages of the child. These lines have been varied a little from the true contemporaneous calcification lines to suit better the apparent positions upon teeth that are shortened in severe cases. This chart will point

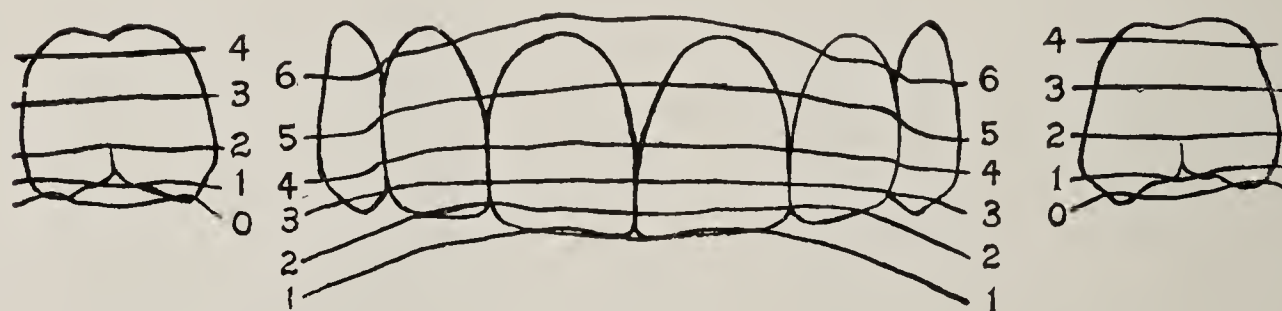


FIG. 79. Outlines of the incisors and cuspids and of the first molars, somewhat enlarged, over which lines are drawn representing average positions at which marks of hypoplasia will occur at the ages named in years by the numerals attached. In these 0 represents birth, 1 one year, 2 two years of age, etc. The rule is that in atrophy occurring before the sixth year the bicuspid and second molars are not marked.

out the age at which any injury occurred as well, perhaps, as it can be done in a chart of this character, which, of course, is founded upon averages. Rather wide variations will occur in the time of the calcification of the teeth of individuals, and also between the several teeth of the same individual. There is certainly as much variation as eighteen months in the time of the beginning of the calcification of the central incisors, and a greater range possibly with all of the other teeth, except the first molars. These latter are perhaps the most constant. But these certainly vary from the twenty-fifth week of uterine life to something near six months after birth.

The chart is intended to give only a general average as to the time of the illness that has caused these injuries.

HISTOLOGICAL CHARACTERISTICS.

In presenting the histological characteristics in this form of dystrophy, it may be stated that all cases, no matter how different their outward appearance, present one plan of departure from the normal arrangement of tissues. The differences are due only to position, the number of zones of injury and in the details of sever-

ity. This plan is inseparably linked with the plan of development of the dental tissues.

Except in the pits that often accompany it, the zones of injury always follow the lines of Retzius very rigidly. In the diagram, Figure 80, the lines of Retzius are made especially prominent to recall distinctly their direction on different parts of the enamel cap of the crown of the tooth. In microscopic observation these are usually clearly seen in some parts of the enamel cap, particularly in central labio-lingual sections. They vary, however, indefinitely in prominence in different sections, and in different parts of the same section. Generally, they do not show clearly in all parts of a section. These lines are the index to the growth of the enamel cap. They are the real lines of accretion and show distinctly the order in which the enamel cap is built up, layer after layer, in its

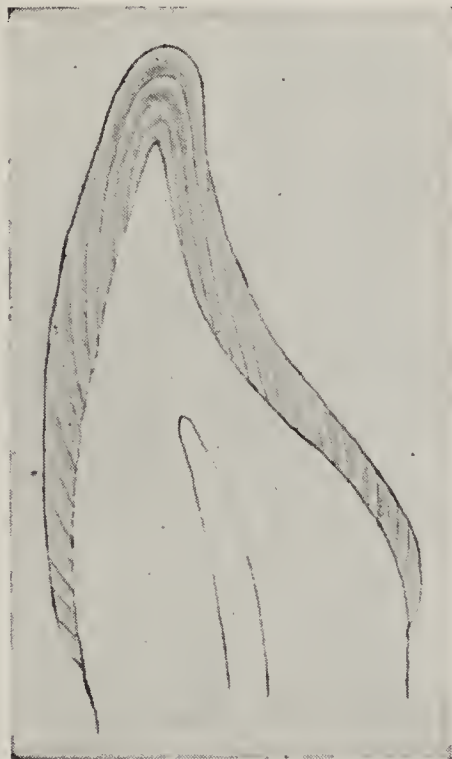


FIG. 80. Diagrammatic illustration of the lines of Retzius, or incremental lines in the growth of the enamel, showing their direction in the different portions of the enamel cap.

growth. This growth begins at the dento-enamel junction, in positions which represent the cutting edge (or points of the cusps in molars and bicuspid) and grows from within outward, while the dentin begins its growth at the same point and progresses from without inward. The growth of dentin is always a little in advance of the enamel as it grows from the incisal edge of the front teeth (or the points of the cusps of other teeth) toward the cemental line.

This contemporaneous accretional deformity, in all cases, consists of an arrest, or partial arrest, of growth of both enamel and dentin in the particular zone being developed at the particular time. In the milder cases growth is imperfect, leaving certain

definite markings outlining the particular parts of the tissue then being formed. In all the severe cases the growth of both enamel and dentin is arrested. There seems to be no recovery of the part of the enamel organ that was at the time in active function. No more enamel whatever is formed over the area affected after recovery from the condition of malnutrition, except as the new formation is telescoped over the area of the old. The dentin pulp, however, rebegins its growth function apparently immediately the condition of malnutrition has passed. But the parts of the tooth which should have been formed during that period are not formed at all. A certain part of the tissue which should have constituted a perfect tooth has been left out, and the distortion of form which we so often see results from patching the second growth onto the first and the total failure of particular portions of the enamel. This total failure of the enamel is not in the direction of the thickness, but is always on the lines of Retzius. Therefore, as we shall see later, there is not a failure of the total thickness of the enamel at any point, except in a few cases in which the injury occurs at a time when calcification was just about to begin, as sometimes occurs in the so-called Hutchinson tooth, and may, rarely, occur in others. For instance, in the diagram, Figure 80, there are four layers of enamel represented over the incisal edge. If total arrest of growth should occur at the time the first two layers are completed, the third and fourth layers will never be formed. The enamel will remain over the incisal edge with only these two layers. Then perhaps the fifth and sixth layers shown, more or less, will also fail, and the seventh and eighth layers will overlap the first and second somewhere near half their length, because the formed part of the incisal edge sinks into the dentin pulp. The dentin pulp has also stopped its growth at the same time and the part that failed of growth is left out of the final tooth form. These are the fundamental propositions presented in the explanation of the histological groupings of tissue and the shortening of the tooth crown found in these cases.

Figure 81 is a photomicrograph of a little more than the incisal half of a crown of a central incisor, showing two zones of injury. Figure 82 shows an entire crown with a single zone of severe injury. In each of these the malnutrition was so severe as to arrest the growth of both enamel and dentin. In each an injury has occurred, affecting the incisal edge of the tooth. By comparing these with the diagram it is easily seen that when a certain thickness of growth of enamel had formed over this part, development was arrested and no more enamel was formed. In each case the enamel is thickest at the incisal edge and thins away to the groove which encircles the tooth crown, which is here presented in section. A band of very dark growth is seen under the

new after-growth of enamel following the lines of Retzius on down to the dento-enamel junction. A comparison with the diagram shows that the growth has been arrested on the lines of accretion or lines of Retzius, Figure 80, in both cases. Also, it is seen that the second injury in Figure 81 is similar in plan to the first, differing in detail only because of the changed direction of the lines of accretion. In Figure 81 the incisal edge is broken, as usually occurs in these thin edges, but Figure 82 is from a tooth extracted soon after it came through the gums and all of the tissue formed is present.



FIG. 81. Section of an incisor shewing two zones of injury.

Figure 83 is an illustration with a much higher power of the labial side of the first zone of injury shown in Figures 81 and 82. Figure 84 is from the second zone of injury on the labial side. In these, the tissues and the lines of Retzius are fairly well shown, and by studying the photomicrographs carefully, the relations of the tissues formed before and after the injury may be made out. It will be noted in Figure 83 that the one particularly dark band, which represents the surface of the enamel formed over the incisal edge, is continued under the enamel of second formation to the

dento-enamel junction. Beginning a little farther from the incisal, a line of interglobular spaces appears in the dentin, and running almost parallel with the dento-enamel junction, continues on toward the incisal edge. Faint traces of these appear even in the small picture, Figure 81. With sufficient amplification, this line of interglobular spaces is found to continue to the incisal edge and join with the similar line from the opposite or lingual side; that is, in the whole tooth it is a sheet or zone of interglobular spaces passing throughout the full extent of the dentin, of which this is a section. This line represents the injury in the dentin. It also represents more. It marks the boundaries of the old and the new formation of dentin and is the line on which these have been

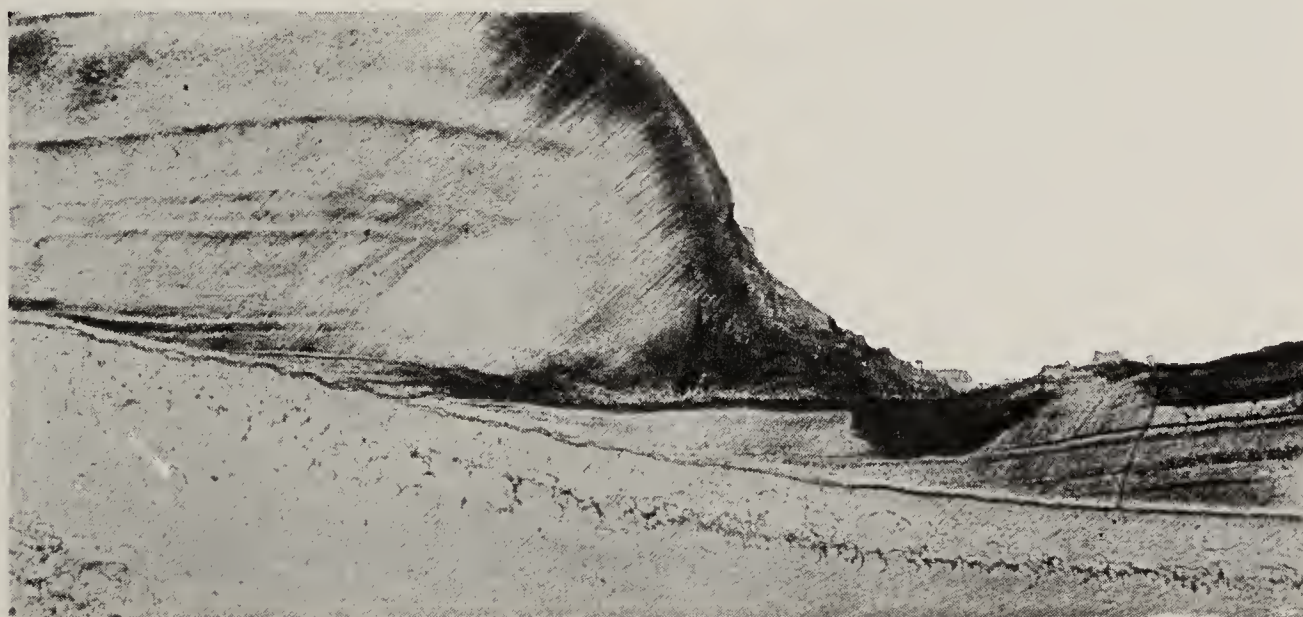


FIG. 82. Section of a cuspid showing a single very severe zone of hypoplasia or a single groove in section. Magnified eight diameters. This case shows well the sinking of the incisal edge into the body of the crown of the tooth.

patched together. On the other hand, the one dark line in the enamel marks the line on which the new formation of enamel is patched onto the old. After a very careful study of sections from many of these teeth, it becomes clear that the part of the tooth which should have formed during the stoppage of growth was not formed at all. The enamel organ was destroyed through its whole thickness to the point where the dark line limiting the first enamel formation reaches the dento-enamel junction, and when the second formation began it was telescoped over the old and laid down upon it, as shown in the illustration. The crown of the tooth was shortened that much, certainly, and may have been shortened very much

more. Figure 82, with its single line of injury, shows how the little part of the incisal edge formed before the injury is literally sunken into that portion formed later, and it is certain that the shortening is much greater than that shown by the apparent telescoping of the parts. In the dentin the same thing occurs, only that it is expressed differently because of the different character of the tissue. The line of interglobular spaces shows where the second growth was telescoped *into* the first.

A study of Figure 84 shows exactly the same plan in the arrangement of the tissue in the second zone of injury, including the overlapping of the new enamel onto the old and the accompanying line of interglobular spaces in the dentin. The shorter overlapping of the enamel at the point of injury is due to the



Incisal End.

FIG. 83. Hypoplasia. A photomicrograph of a portion including the zone of injury nearest the incisal edge on the labial from the same section shown in Figure 81. In this the lines of Retzius may be seen in the enamel, also the dark line of junction between the enamel of first formation and enamel of second formation, reaching from the dento-enamel junction, with the enamel of second formation overlapping that of the first. The line of interglobular spaces in the dentin running almost parallel with the line of the dento-enamel junction, is well shown.

changed direction of the lines of growth. When we study the short and stumpy forms of many of the crowns of these malformed teeth, we must conclude that the shortening is often very much greater than this overlapping. It is this shortening and telescoping together of the different parts that is responsible for the greater part of the distortion of form so often observed in these teeth. It appears certain that the tendency is to form each of the parts on the lines that each would have had at the specified time of growth if there had been no interruption of the growth.

Another view of this may be gained by studying the lines of the labial dento-enamel junction. In the study of sections of many human incisor and cuspid teeth, this line is found to form a con-

tinuous curve from the incisal edge to the gingival line, as seen in the diagram, Figure 80. The amount of curve may vary indefinitely, but it is always a continuous curve in every normal tooth. In sections of these malformed teeth, this curve is found broken by a recurve at the zone of injury in every case, even in the lighter forms in which the growth seems not to have been completely arrested. This disturbance of the direction of this line seems to be due to the effort to form the second part on the lines that would have been laid down at that time if the growth had been going on regularly, and the larger and smaller are patched together. In the dentin the growth has been in abeyance and the growth begins on the lines on which it was left off. But immediately the tendency is to enlarge to the greater outline of the tooth as it would have



FIG. 84. Hypoplasia. A portion including the second zone of injury seen in Figure 81. In this position the lines of Retzius diverge more sharply from the direction of the line of the dento-enamel junction, and the overlapping of the third growth of enamel onto the second is shorter. The discoloration is greater. The line of interglobular space is broader, and in this position diverges more sharply from the line of the dento-enamel junction. Otherwise it is similar in plan with the first zone of injury shown in Figure 83.

been at the time had the growth not been interrupted. This causes a recurve in the line of the dento-enamel junction. In the enamel, the telescoping seems to be actual. That part of the enamel organ that had not arrived at the period of enamel building is uninjured and is pushed forward over the previously formed enamel and lays down its layers of the second growth of enamel thus far over the old. No other explanation of the phenomena is presented after the study of numerous cases.

The discoloration that occurs in these teeth would seem to be an essential characteristic, if it were judged entirely by the teeth obtained for making sections. This material is very difficult to find. Only extracted teeth can be used, of course, and few of them are extracted until so badly decayed that they are useless, except

those that are so badly discolored that patients and their friends urge their removal on that account. Examinations in the mouth reveal many cases of very considerable deformity without notable discoloration, as the photograph, Figure 75, taken from the mouth, attests. Many of the zones of injury show no discoloration.

Numerous writers have given short descriptions of these teeth, scattering back for more than a hundred years. Most of these have dealt with the outward appearance only. Very few have published studies of the histological characters, and most of these have been very brief and imperfect. Among the better should be mentioned Wedl, 1870; Baume, 1882; Walkoff, 1885. But by far the most important of the studies is that by Dr. Otto Zsigmondy, of Vienna, Austria, in a paper presented at the World's Columbian Dental Congress in Chicago in 1893. Unfortunately

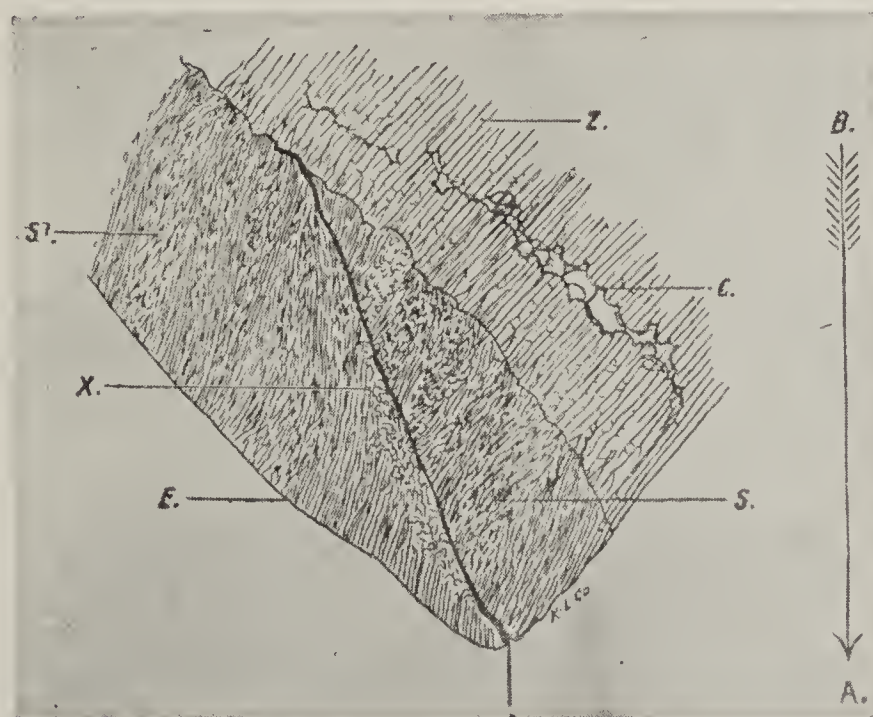


FIG. 85. Lengthwise section through portion of crown of an hypoplastic tooth. A. Direction toward the crown. B. Direction toward the root. Z. Dentin. C. Interglobular spaces. E. Enamel. S. Wedge-shaped piece separated by line of injury. X. Line of injury. S¹. Full thickness of enamel. *Zsigmondy*.

for Americans, no translation into English has been published. The author examined many of Dr. Zsigmondy's sections and learned further of his conclusions in conversation. It was his conviction that the tissue distortion was produced by a condition that had been of very short duration, because the apparent zones of injury in the dentin were often — nearly always, indeed — so very narrow when considered in their relation to the developmental lines. He could not, therefore, account for the marked deformity of these teeth. At the time he wrote he did not have the advantage of photomicrographic reproductions, and his illustrations were very meager and insufficient. One of the best of them is reproduced in Figure 85.

Figure 88 is a photomicrograph of a section of the labial portion of a zone of injury of the milder sort apparently, occurring in a central incisor. In this there was considerable discoloration of the enamel occurring irregularly along the line of injury in the

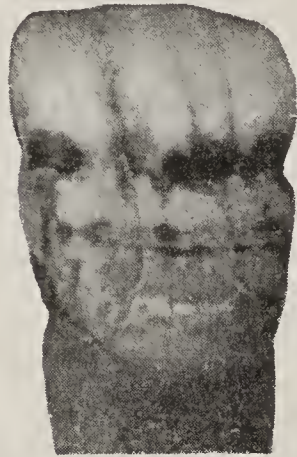
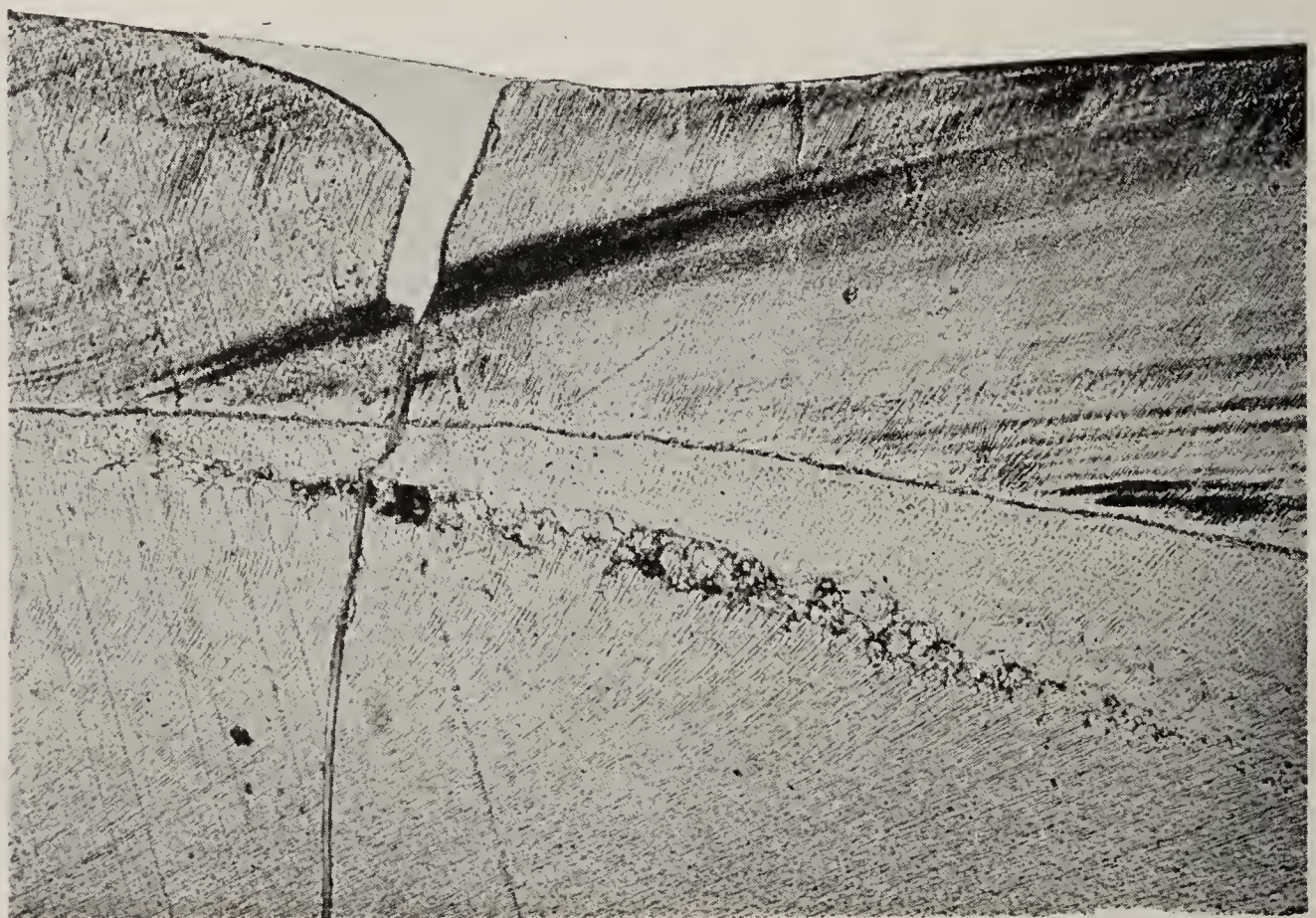


FIG. 86.



FIG. 87.

FIGS. 86 and 87. Labial and lingual surfaces of a central incisor, photographed to show the appearance of the groove. The dark color in parts of the groove makes it appear deeper than it really is. A part of a section cut from the labial side of this tooth is shown in Figure 88.



Incisal end.

FIG. 88. A photomicrograph of a portion of a section from labial portion of the central incisor, Figures 86, 87, showing a milder sort of injury, in which the growth of the enamel was interrupted but not permanently stopped. The line of interglobular spaces literally divides the dentin of first formation from that of the second. The section was broken and the parts placed in position. A piece of enamel was lost in preparation, as represented by the dotted line.

labial surface, as shown in the photograph of the tooth. Figures 86 and 87. The discoloration in the line of the groove has the effect of a shadow in the photographs and makes the groove appear deeper in the discolored portions, which is not the fact. The par-

ticular section from which Figure 88 was made was chosen from a part showing the least discoloration. In this case the only distortion of the crown apparent in a superficial view of the tooth is the groove encircling the tooth and the discoloration. Also, the section shows that there was not a complete arrest of growth of the enamel. With a good light the enamel rods may be traced with the microscope through the darkest lines of the section, and they are seen to be well formed. There is no appearance of the telescoping process. The groove in the enamel appears much less pronounced in the section when highly magnified as shown, than it does in the photographs of the tooth. In the dentin, however, the injury is very severe, as shown by the clean-cut continuous line of interglobular spaces, which literally cut the dentin first formed

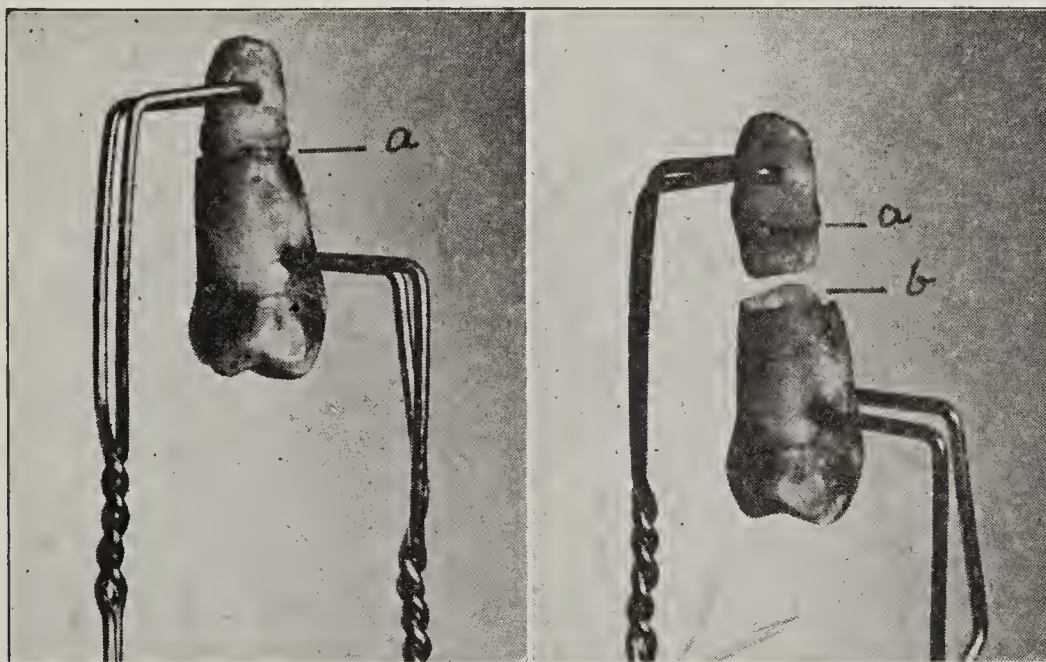


FIG. 89.

FIG. 90.

FIGS. 89, 90. Root of tooth parted on lines of growth. Photographed from the specimen extracted by the author. Photographs of a bicuspid tooth which had a zone of injury mid-length of the root, and which was pulled apart in telescope form along the line of injury, i. e., the line of interglobular spaces. In Figure 89 the parts are photographed in normal position. In Figure 90 the the two parts are separated, showing how they are telescoped together.

from that formed later, and in the examination of the labial line of the dento-enamel junction in the full section, it is found to be distorted by a recurve, showing the interference with growth to have been profound and that some real shortening of the tooth must have occurred.

As a further illustration of the possibilities in this class of injuries, two photographs, Figures 89 and 90, are presented of a bicuspid tooth showing the separation of the telescoped parts in an injury of this kind occurring mid-length of the root. The author extracted this tooth. The patient, a stranger, applied for relief from caries of bone of the upper jaw, and this tooth was situated on the border of the carious area in such position that it seemed best to remove it, though the tooth and its individual alveolar process were otherwise in good condition. At the moment

of removing the tooth, it was noticed that the apical portion of the root did not come away, but was pulled from its place and remained loose in the alveolus. Laying the tooth on the bracket with the forceps, this apical portion was picked out with the foil pliers and laid with the tooth for after examination, because it seemed to be a very curious break. The operation was completed and the patient dismissed with an appointment to return later for further treatment. On examination, this tooth and root were found to have pulled apart like a telescope tube, and the telescoping was on the lines of growth of the dentin. Figure 89 shows the tooth and root placed together in the normal form, in which *a* marks the line of break. In Figure 90 the two parts are separated, showing how



FIG. 91. A portion of a section through a central incisor showing hypoplasia of the severer sort. The plan of the injury, and the telescoping together of the parts, is seen to be the same as in Figures 83, 84, but the discoloration is much less. The thin incisal edge had been ground away before extraction in an effort to improve the appearance.

the apical portion telescopes into the body of the root. There must have been a severe illness of short duration at the time this part of the root was developing, which prevented the deposit of calcium salts, and a sharp, distinct and continuous line of interglobular spaces occurred. At the time, the root was developed only as far as shown in the lower section of Figure 90, and had the broad conical opening shown at *b*. The internal diameter at the point to which the end of the apical portion reaches was of the size shown by the end of that piece. The result was that the solid dentin formed at that time represented only the lower square end of the upper piece.

This was broken in the effort to extract and the root pulled apart on the line of the area of interglobular spaces, the line representing the lines of the process of growth.

The patient failed to keep his appointment for further treatment and was not seen again. The opportunity to inquire into the nature of the nutritional disturbance that had caused this rare form of injury was lost. The specimen, however, tells its own story clearly. This case shows that the root of a tooth may also be injured by a condition of malnutrition, though such an extreme occurrence as this must be rare.



FIG. 92.



FIG. 93.

FIG. 92. Photograph of a bicuspid, showing imperfectly a slight groove from atrophy near the junction of the middle and gingival thirds of the crown. See Figures 93, 94.

FIG. 93. The bicuspid shown in Figure 92 split mesio-distally and the cut surface photographed as an opaque object. Note a broad zone of shadow in the dentin, extending in a semi-circular form from the groove on the mesial to the groove on the distal side. See also Figure 95.

A considerable number of cases have been observed in which a zone of injury occurred in the dentin beginning below the cemental line, as in the case shown in Figures 89, 90, though they are far more rare than those occurring in the crown of the tooth.

Figure 91 represents another case of injury of the graver sort, occurring in a central incisor, in which but little discoloration is apparent. The wide overlapping of the new onto the older enamel, the complete breakage of the enamel rods along the line of junction of the two, the change in the course of the enamel rods in the two formations of enamel and the profound disturbance of and re-curving of the labial dento-enamel junction, all point to a long suspension of nutrition and account for the grave distortion of the form of the tooth. This is much like that shown in Figure 82.

The line of interglobular spaces is sharp and severe, but very narrow, and the dentin is normal immediately on either side. The incisal edge had been ground away in the endeavor to improve the appearance before the tooth, with the other incisors, was extracted. About one-third of the normal length of the crown had been missing.

The next case is very curious in several respects. It is a bicuspid tooth that showed a very slight groove in the enamel not far from the gingival line. It was covered by the overlapping gum margin, except on the buccal surface. The whole tooth was, perhaps, as white as any normal tooth and was without any discoloration along the line of the groove, except that caused by a deposit



FIG. 94. A photomicrograph from a portion of a section of the bicuspid shown in Figures 92, 93, showing zone of shadow in the dentin as a result of interference with nutrition. Markings of this character are found in the mildest forms of interference with nutrition that show atrophy marks.

of dark, closely adherent serual calculus at several points. A photograph of this groove was but a partial success, as is shown in Figure 92. The tooth was then divided mesio-distally, preparatory to grinding sections. In examining the halves with a pocket lens, a curious zone of injury in the dentin was discovered, which was photographed at once as an opaque object, which is represented in Figure 93. Two sections, two thousandths of an inch thick, were prepared and mounted without removing them from the cover glass on which they were ground. The sections were beautiful. No one would suspect that there was any zone of injury in either dentin

or enamel. The disturbance of the line of the dento-enamel junction and in the one section a clinging bit of serumal calculus were the only abnormalities discoverable by microscopic examination. The only way this could be explained was that the something that had been seen and photographed had become obscured by the balsam. The balsam was dissolved out and the section dried. A zone of fine interglobular spaces was then found with another singular appearance in the form of a broad line of demarcation, that could not be explained. The section was remounted in a very stiff balsam without using anything to clear the dentin, with the expectation of making a photomicrograph the same evening. Something prevented, and by the next evening, the day having been unusually warm, the interglobular spaces were again filled with

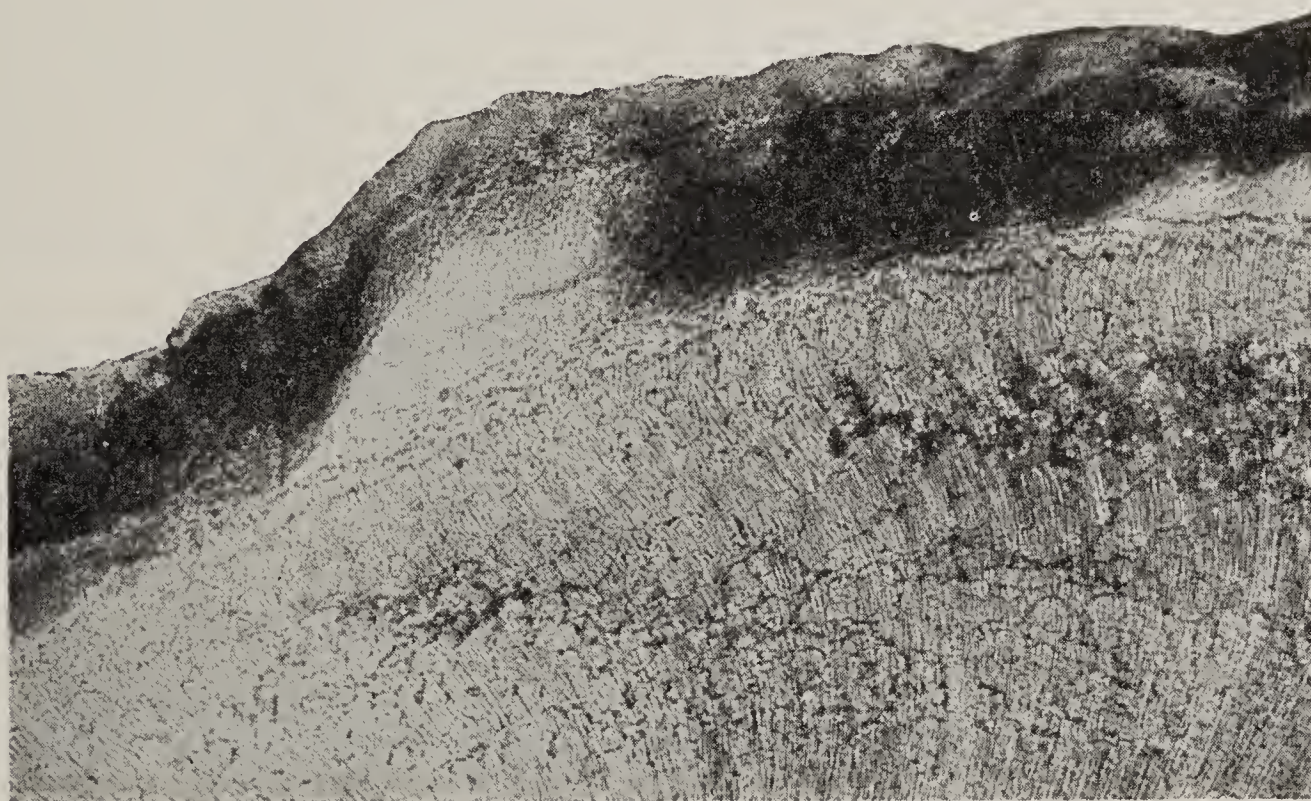


FIG. 95. A photomicrograph from section of hypoplastic tooth cut diagonally to the zone of injury and serving to exaggerate the width of the zone of interglobular spaces in the dentin. This gives a stronger representation of the real injury to the dentin.

balsam. The shadow, however, remained, and is presented in Figure 94. It has since been found that the condition presented is common to a considerable number of the slighter injuries of this type.

Figure 95 is a photomicrograph of a labio-lingual section cut from near the mesial side of a malformed tooth so that the line of interglobular spaces is cut through diagonally. This gives an exaggerated view of the zone of injury to the dentin, but will serve to impress the fact that these injuries are very severe.

This presents this subject from its gravest to its slightest degree, in sufficient variety of cases to render the conditions intelligible.

THE DEFORMITY IN THE FIRST PERMANENT MOLARS.

The deformity of the first permanent molars should receive special consideration because of its greater frequency and because it so generally leads to early and rapid caries beginning in the malformed portion. The plan of injury does not differ from similar deformities in the front teeth, but the details of the injury are different because of the wide difference in the form of the tooth. Greater frequency of the occurrence of the condition in these teeth is due to the earlier beginning of calcification. In dissections of the jaws of the fetus at term the calcification of this tooth is usually found to have just begun on the points of the cusps. Sometimes there are only small spiculæ, in other cases a more considerable part of the cusps is calcified, but the calcification has not been observed to be so advanced at birth that the cusps were united by calcified tissue, and evidently they are not so united until much later. On the other hand, it was only occasionally that the least

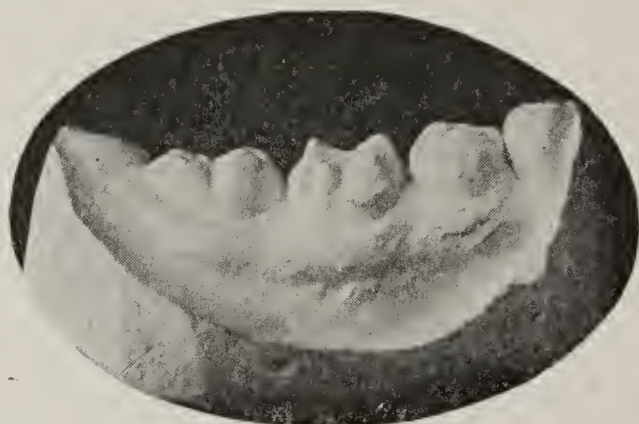


FIG. 96. Photograph of cast showing hypoplasia of a first molar. The occlusal portion of the tooth is much reduced in size. This is best appreciated by comparison with the second molar. Normally the occlusal surface of the first molar is the larger of the two.

bit of calcification had occurred on the central incisors. More often calcification does not begin on these until about the end of the first year. Therefore, an illness that brings about serious malnutrition during the first year of the child's life is liable to wreck the occlusal surfaces of the first molars, while all of the other teeth escape injury.

The injury to these teeth occurring so early is very characteristic if seen soon after the eruption of the teeth and before further injury has occurred by breaking away the sharp spiculæ representing the malformed cusps or by caries. But it is exceedingly difficult to obtain specimens from which to make illustrations. If the injury has occurred very early, or before the enamel plates forming the lobes of the teeth have joined together, the spiculæ of cusps will stand much closer together than the cusps of the normal tooth. The whole of the occlusal surface of the tooth is



FIG. 97. A photomicrograph from a perpendicular bucco-lingual section through the crown of an atrophied lower first molar. The injury to the enamel is slight as compared with the injury to the dentin. Notwithstanding this, the shortening of the crown has been very great, as will be seen by the short distance from the occlusal surface of the tooth to the pulp. This is a common characteristic of these teeth. The line of interglobular spaces in the dentin follows close to the dento-enamel junction under the whole of the occlusal surface. It then dips down along the axial surfaces, as shown at both the lingual and buccal margins of this section. Through most of its course this appears as a sharp, but jagged, dark line of interglobular spaces, which are better shown with the great amplification in Figure 98. All of this dark part of the line is made up of interglobular spaces that open the one into the other. These are filled with air when the section is dried. At several points the balsam used in mounting the section has run through this, partially obscuring it.



FIG. 98. A photomicrograph with a much higher power from another tooth with a similar atrophy to that shown in Figure 97, in which the section was cut horizontally or crosswise. The section through the interglobular spaces is, therefore, through that portion of the line showing in Figure 97, which dips toward the gingival at the buccal and lingual portions of the section. E. Enamel. D. Dentin. S. S. Line of interglobular spaces appearing as irregular dark blotches connecting the one with the other in every direction.

dwarfed, often discolored and sunken into the tooth crown. The pulp chamber will be nearer to the occlusal surface than in the normally developed tooth in proportion to the sinking of this part into the body of the crown. Figure 96. All around this the enamel of second formation wells out in the form of a broad collar to the normal size of the tooth crown at or about the base of what would have been the normally developed cusps. The whole of the injured area is apt to be rough and pitted, and the pits and grooves are very apt to be wide open. This condition gives unusual opportunity for the beginning of caries and the early exposure of the

pulp. It is for this reason particularly that they are so often destroyed very early. In cases occurring a little later, but before the completion of the occlusal surface, the conditions inviting the beginning of caries are equally bad. The cusps will stand further apart, are stronger, but the central part of the occlusal surface and the outer slopes of the cusps are in very bad shape. At a still later date, after the completion of the occlusal surface, the injury



FIG. 99. A photomicrograph from a portion of mesio-distal section of a first molar showing in the section two zones of interglobular spaces. It shows only a part of the mesial half of the section. Note that the line of interglobular spaces nearest the enamel, the first line, follows closely the dento-enamel junction. This follows the prominence of the mesial marginal ridge of dentin (on the left in the picture) and then dips down to the gingival. The line of injury to the enamel also rounds over this prominence and again touches the dentin just above the point of ending of the injury to the dentin. The second, and more marked zone of injury to the dentin, swings out quickly from the dento-enamel junction, on the left in the picture, and runs across much deeper in the tooth, showing the amount of dentin formed before this occurred.

appears as a groove around the crown of the tooth and is generally of less consequence.

In all of these cases the zone of injury in the dentin is one of the very grave features, for the reason that caries reaching this zone of interglobular spaces spreads through it quickly. It has been exceedingly difficult to get material for the illustration of this for the reason that very generally the occlusal surfaces of these teeth are destroyed by caries before their removal, rendering them useless for this purpose. Figures 97, 98 and 99, with their descriptions, serve, however, to illustrate the condition of the tissue injuries fairly well, though neither of them are of the severer

forms. Figure 97 particularly shows the line of interglobular spaces in the dentin extending across the occlusal surface and dipping down gingivally along the axial portions of the dento-enamel junction. This figure also shows well the shortening of the crown between the pulp of the tooth and the occlusal surface. From the position of these zones of injury, it will be realized, from a careful study of the lines of the injury to the dentin, that the total shortening of the crown of the tooth is between the pulp and the occlusal surface in the molars. It is also between the pulp and the incisal edge, where it occurs in the incisors and cuspids. This is the reason that pulps are so frequently exposed in the preparation of cavities in these teeth. Dentists generally have not understood that the pulps were so close to the surface. When it is realized that these sheets of interglobular spaces, of which the zone of injury in the dentin showing in Figure 97 and in Figure 99 is a section spread through the entire area of the crown of the tooth, forming openings through which microorganisms may readily grow, it will be understood why it is so difficult to prevent the destruction of these teeth by caries. It will also be understood how decay may quickly undermine the entire enamel cap, allowing it to fall away, exposing a blackened stump of dentin in all of the central portion with jagged enamel upon its margins, with decay persisting around the circumference where the line, or sheet, of interglobular spaces dips to the gingival near the dento-enamel junction. In the child, one is prohibited from forming a cavity of such depth as required to hold such a broad restoration because of the nearness of the pulp of the tooth.

The grave significance of this lies in the fact that caries beginning in the imperfections of the occlusal surface quickly reaches this zone of interglobular spaces and spreads rapidly through it, undermining and destroying the whole occlusal portion of the tooth. It often happens that the whole of this has been swept away so quickly that the area is uncovered before decay has proceeded further toward the pulp. The decaying area is then fully exposed to the fluids of the mouth, and the progress of the caries is arrested. In this case, the tooth remains as a blackened stump that soon rises in its alveolus and occludes with its fellow, which is generally in a similar condition, and both do good service. More often, however, decay continues in that portion of the zone of injury referred to, that dips under the strong enamel around the margins of the crown. Being thus protected, it continues to burrow, finally reaching the pulp and completing the destruction. This is the general fate of these malformed first permanent molars.

TREATMENT.

FIRST MOLARS. Treatment of these malformed first molars to prevent the results detailed above, is exceedingly desirable. The

treatment is required as early as the eighth year, often in the seventh. Restorations may be successfully placed in a few cases. In cases in which the child can be controlled to do this work, the fissures should be properly prepared and restored with gold at once when discovered. Generally, however, it will be found impracticable to make the proper preparation for restorations at so early an age because of failure to control the child. As a rule it would be necessary to do this during the seventh or eighth year. A large proportion of these cases are hopelessly decayed before the ninth year, and a considerable number are decayed to exposure of the pulp, or the occlusal surface is lost during the seventh or eighth year. Ordinarily they will be seen first by the dentist when the child is brought for consultation regarding the deformity of the incisor teeth, the parents not having noticed the deformity of the first molars. Even at that time, in many cases, the first molars will be found badly decayed. This very early appearance of caries in these cases, and the fact that the pulp of the tooth is so often exposed by a decay that seems not to be very deep, greatly increases the difficulty of treatment.

When the teeth can be seen very early, or as soon as they have come through the gums, and the occlusal surfaces are found badly deformed, showing many wrinkles and deep fissures, it is generally best to grind away any small, sharp spiculæ of cusps that are liable to be broken in chewing food. Then, if decay has not actually begun in the fissures, these may be dried out and restored at once with copper cement without further preparation. Often such a course will be necessary in order to do anything that will be of service to the child. In the deeper fissures in which decay generally starts earliest, it will do excellent temporary service. When decay has made some progress the softened material should be removed, after breaking away any undermined enamel, and a restoration should be made with copper cement. These should be examined every three months to see that they are doing well and to make replacements if indicated. In this way these teeth can often be tided along and serious decay prevented until such time as permanent operations can be made.

Those cases in which the condition of the occlusal surface is still worse and in which decay seems to start in spite of the effort to prevent it in this way, a gold cap may be made to cover in the entire exposed part of the crown. After grinding down the more prominent points that will cause the cap to interfere with the occlusion, an impression should be taken in modeling compound. From this a gold cap can be formed to cover in the occlusal surface of the tooth and may be cemented in place. There should be no attempt to make a full gold shell crown. It should only overlap the axial surfaces sufficiently to hold it in place. A renewal later, when it can be slipped further over the tooth, should be expected

in many of the cases. The gum will cover much of the crown at the age of seven or eight years, and the child will be very sensitive about any pushing away of the soft tissues.

Sometimes it will be impossible to place these without some interference with the occlusion. This will not be of much importance if the interference is not very considerable, for the occlusion is quickly accommodated by movement of the teeth in early childhood. Special attention should be given to the intercusping of these caps that the teeth may not be caused to slide out of position during the process of shedding the temporary teeth. Generally the crowns will have to be made rather flat and the cusps short in order to prevent raising the bite excessively, especially if this must be done after the teeth have come into full occlusion.

These caps should be regarded as a temporary expedient. When the child is older the time will come when the teeth may be permanently restored. In the bad cases, this should not be attempted before the person is eighteen or twenty years old. The pulp is so near the occlusal surface that it will be much endangered if a restoration with sufficient anchorage be undertaken earlier. In the meantime a careful guard must be kept to see that decay does not begin on the axial surfaces along the gingival margins of these caps. Caps put on in this way at from seven to ten years of age, even if they reach fully under the free margin of the gum at the time they are placed, will be some distance from the free margin of the gum after a few years. If decay occurs, a new cap may be made to cover it.

It is the duty of every dentist having families in his charge to see to it that these malformed molars are taken care of in this way, or some similar way, very early. After the patient has grown older and the teeth have come further through the gum, other devices may be used if thought necessary.

Some cases may be successfully treated by grinding away a considerable part of the injured enamel and thus gain a smooth surface that will be kept clean by the excursions of food in mastication. This may often be employed advantageously after considerable decay has occurred. For little children it should be done little by little, having them come to the office frequently for this purpose. Particularly this should be the case if any sensitiveness has developed.

INCISORS AND CUSPIDS. The treatment of incisors and cuspids should be along different lines. In a very large proportion of these cases, no treatment whatever is necessary, for these teeth are not much inclined to decay because of the injury. Sometimes decay does occur, and if the injury is confined pretty closely to the incisal edge, it may usually be treated by grinding away. Often the injured portion may be ground away so that the tooth will look fairly well.

The teeth will be a little bit short, but the inclination will be to protrude farther through the gums and increase their length in that way. If necessary, other teeth in the neighborhood may be ground also, shortening them a little, and in this way the esthetic requirements may be satisfied, removing much, sometimes all, of the injured portion. Often teeth that are marked by grooves may be made to look very well by grinding the enamel smooth and level, making in this way a fairly good tooth form, being careful, however, not to expose the dentin on the labial surface. Exposure of the dentin on the cutting edge is not very objectionable.

Frequently much harm is done to these malformed teeth by attempting to fill blackened pits. At the points where these occur, the teeth are often very thin and an injudicious effort to excavate results in cutting through to the lingual surface, and after removing perhaps considerable material to gain the proper anchorage, the operator finds the incisal portion of the tooth too weak to stand. No restoration should be made for the purpose of closing up blackened pits, unless it is first found that there is abundant tooth material for strength after the excavation has been made. Also it must be remembered that the pulps of these malformed incisors are very near the incisal edge as a rule, and exposure of this organ in the excavation is very liable to occur.

Incisor teeth that are so malformed as to be very unsightly in appearance, should not be treated hurriedly. They should be tided along until the time when porcelain jacket crowns may be made. If they can be kept in position without other treatment than that intended as temporary, until the patient is eighteen to twenty years old, it is very much better that it should be done. Certainly no crowning operations should be undertaken in these teeth before the patient is sixteen years old, and eighteen to twenty is very much better.

Long observation of pulp removal and the filling of root canals for young people shows that these teeth do not do well. It is true that the pulps can be removed and the canals filled of the central incisors at twelve years of age in very many cases with results that, within the first year or two, seem perfect, but it is also true that these teeth tend to break down, the roots to split, or some catastrophe is very likely to happen to them before the patient is twenty-five years old, and the teeth are lost.

Many of them suppurate after they have been apparently in perfect health for several years after the root filling has been made. All of these considerations unite to indicate that the removal of the enamel in order to place porcelain jacket crowns be delayed as long as possible. The roots of these teeth are just as good for the purpose of artificial crowns as the roots of fully developed teeth; in fact, all of that portion of the tooth root-wise of the injured part is normally developed as a rule.

THE ENAMEL WHORL.

Pits are a common accompaniment of hypoplasia, but in no way a necessary part of it, as is shown by the many cases of even the severer injuries in which they are absent. Enamel pits seem not to occur in more than one-fourth of them. Further, these enamel pits occur in teeth that are otherwise normal. It is not very rare to find a single pit in the enamel of some one tooth of an otherwise perfect set of teeth. In microscopic sections, the same histological characters are found as in pits accompanying the contem-

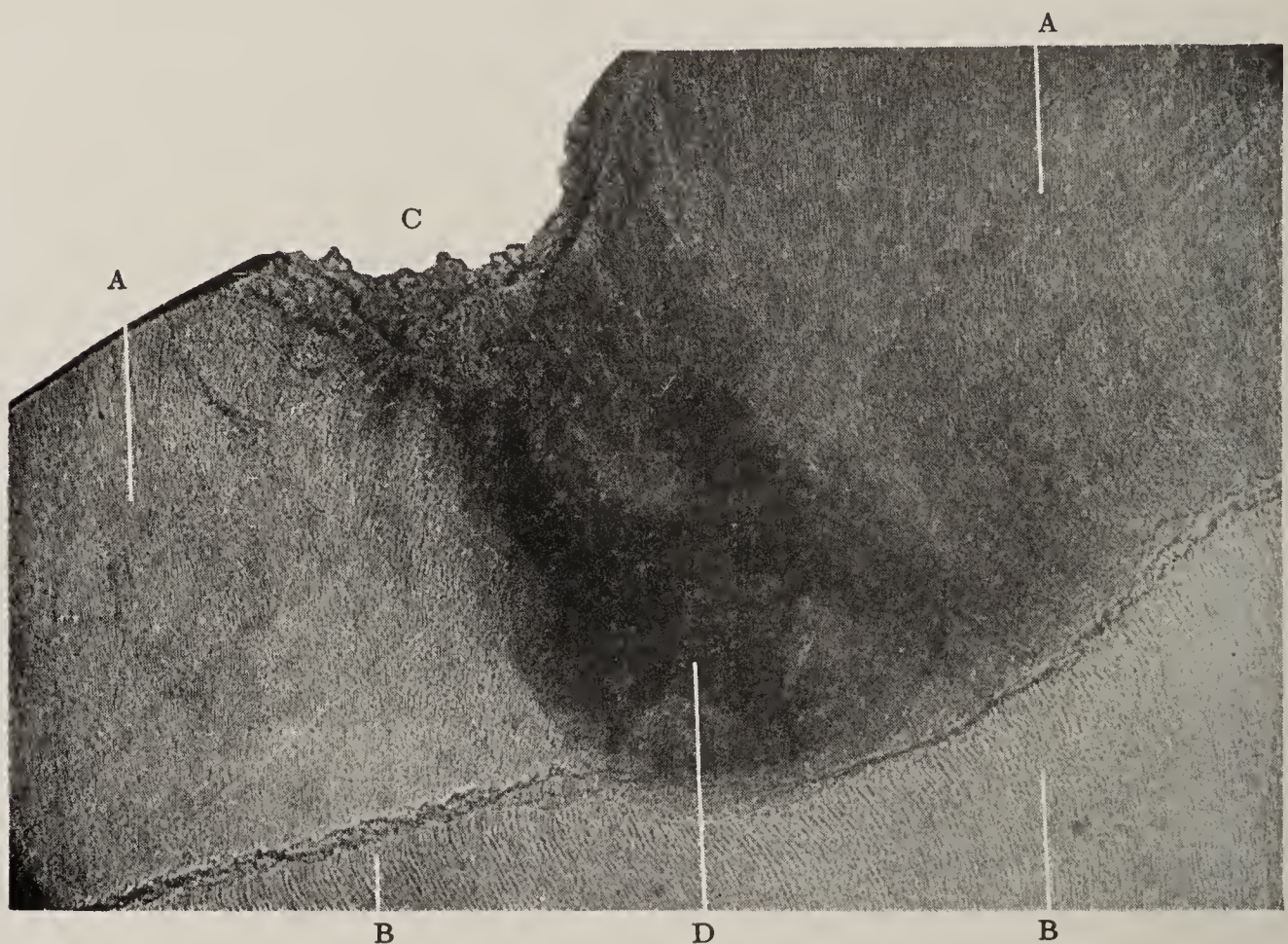


FIG. 100. An enamel whorl in the enamel near the dento-enamel junction, the surface of the enamel presenting a pit over the whorl. Normal enamel is marked A, A; dentin, B, B; the pit in the surface, C; the whorl, D. It will be observed that there is a heavy deposit of brownin in the deformed enamel, which lies next to the dentin. In fact, there is a recurve of the dento-enamel junction to partially accommodate the whorl.

A little different direction in the cutting of the section, so that the pit in the surface would be missed, would show only the dark spot in the enamel and the whorl in the direction of the enamel rods, which would appear if the spot was not so black as to interfere with seeing the enamel rods.

poraneous accretional deformity. They mark an imperfection, or partial failure, in the development of the enamel rods, confined to a small area, usually round, and evidently are most apt to occur in cases where there has been some marked difficulty in enamel development. Hence, they are a very common accompaniment of the hypoplasia. Not infrequently, the rows of pits in the enamel are the only signs of injury to the teeth as a result of an illness. In this case the rows of pits form zones on the parts of the teeth contemporaneous in development, as one of the expressions of the accretional deformity. Aside from conditions of general malnu-

trition, pits may occur in any part of the enamel, showing no especial preference as to teeth or locality on any tooth.

The pit marks the failure of development of the enamel rods at a point, leaving a hole of more or less depth. This is generally filled, or partly filled, with an amorphous material, dark in color, varying from a yellowish hue to a deep black. In such cases, the



FIG. 101. A photomicrograph of an enamel whorl beginning in the midst of enamel tissues, showing a failure of the development of enamel rods. The pit is nearly filled with amorphous material, very dark in color, and much of the enamel in the neighborhood is discolored.

dentin appears never to be exposed although some of the pits are as deep or deeper than the normal thickness of the enamel. In all of these cases of very deep pits there is a depression in the dento-enamel junction, as shown in Figure 100, and a lining of enamel, in which the enamel rods are arranged in a segment of a whorl, all pointing to the center like the spokes of a wheel, in the bottom

of the pit. When the enamel rods forming these whorls have grown about a certain length, growth ceases, and an opening is left, and this may extend as an open pit to the outer surface of the enamel, leaving an opening the full depth. Or this may be filled in part by a dark material not resembling enamel. Or, again, the surrounding enamel rods may close over it, partially or completely obscuring the pit, so as to form a smooth enamel surface over it. In Dr. Callow's case, described later, the deformity consists mostly in numerous whorls, many of them extending deeply into the dentin. These definite whorls are at the dento-enamel junction, or actually partly within the dentin. But many pits are formed within the usual thickness of the enamel without disturbance of the line of the dento-enamel junction, as shown in Figure 101. In most of



FIG. 102. This photograph is from a skull found in the anatomical laboratory of the dental department of Creighton University by Dr. E. H. Bruening. All of the teeth of this individual presented the same deformity as those shown in the illustration.

A section prepared from this skull was lost by accident. The scalloping was very regular. In this case the teeth presented an irregular wrinkling upon their surfaces, the wrinkles passing horizontally around the teeth. These wrinkled teeth have always a scalloping of the dento-enamel junction.

these cases there is much disturbance of the direction of the enamel rods in the immediate neighborhood as the rule. In some there seems to be a failure of the enamel organ to perform its function of rod formation at the particular spot. In and about many of these pits there is deep discoloration. Many times the dark color is confined to the pit itself, which is partially filled with a substance strongly resembling the chitinous covering of insects in appearance and in which no enamel rods can be made out. In other cases the dark color extends broadly in, or among, the enamel rods about the pit.

In these pits the disturbance follows the general direction of the enamel rods in the locality. This is as true of the pits that

accompany the accretional deformity as those that occur as isolated defects. In this the pit is distinctly different from the other injuries to the enamel in hypoplasia, for these as uniformly follow the lines of Retzius.

The pathology concerned in the formation of these pits is obscure. Their prevalence in cases of hypoplasia indicates that they are due to disturbances of nutrition. They consist essentially in a failure in the formation of enamel rods. But such remarkable disturbances as those shown in Dr. Callow's case indicate that there may be some other pathological element not yet understood. Histologically, they seem to be a very distinct form of dystrophy. The rule is that these pits need no treatment. They rarely decay, but as there is a slight opening and the tissue is often black or dark



FIG. 103. A cuspid, a bicuspid and a molar tooth from Doctor Callow's case. These are fair representatives of the appearance of the other teeth from the same mouth, from several of which sections were cut. An extreme case of wrinkled teeth.

colored for a little distance around it, dentists frequently cut them out and make restorations. There is no reason for doing this unless softening has occurred, or in other words, unless decay has actually begun. Of course in that case the restoration is the proper procedure, but not otherwise.

CORRUGATED TEETH.

Teeth presenting an unusual dystrophy, to which the term corrugated teeth has been applied, are characterized by an irregularity in the surface of the enamel, and of the line of the dento-enamel junction. The most typical form is shown in Figure 102, in which all of the teeth of the denture presented an enamel surface of alternate ridges and furrows. This is a photograph of a skull found in the anatomical laboratory of the Dental Department of Creighton University by Dr. E. H. Bruening. This defect has no relationship whatever to a definite period of malnutrition, because it does not follow the lines of accretion, and is present in all of the teeth. In

such cases the dento-enamel junction also shows much variation from the normal continuous curve, being wavy and in some cases very irregular. In the case illustrated in Figure 102 the dento-enamel junction was a series of quite uniform scallops.

Several illustrations of another case of this type of dystrophy are shown, Figures 103, 104 and 105. Dr. J. E. Callow of Antigo, Wisconsin, removed sixteen teeth for a young woman who applied to him for treatment. They included incisors, cuspids, bicuspid and molars. The condition of these teeth, as indicated by their outward appearance, is very fairly shown in the photograph, Figure 103, of a cuspid, bicuspid and molar. All of the others were similar. Examination of these teeth showed that the injury to, or the

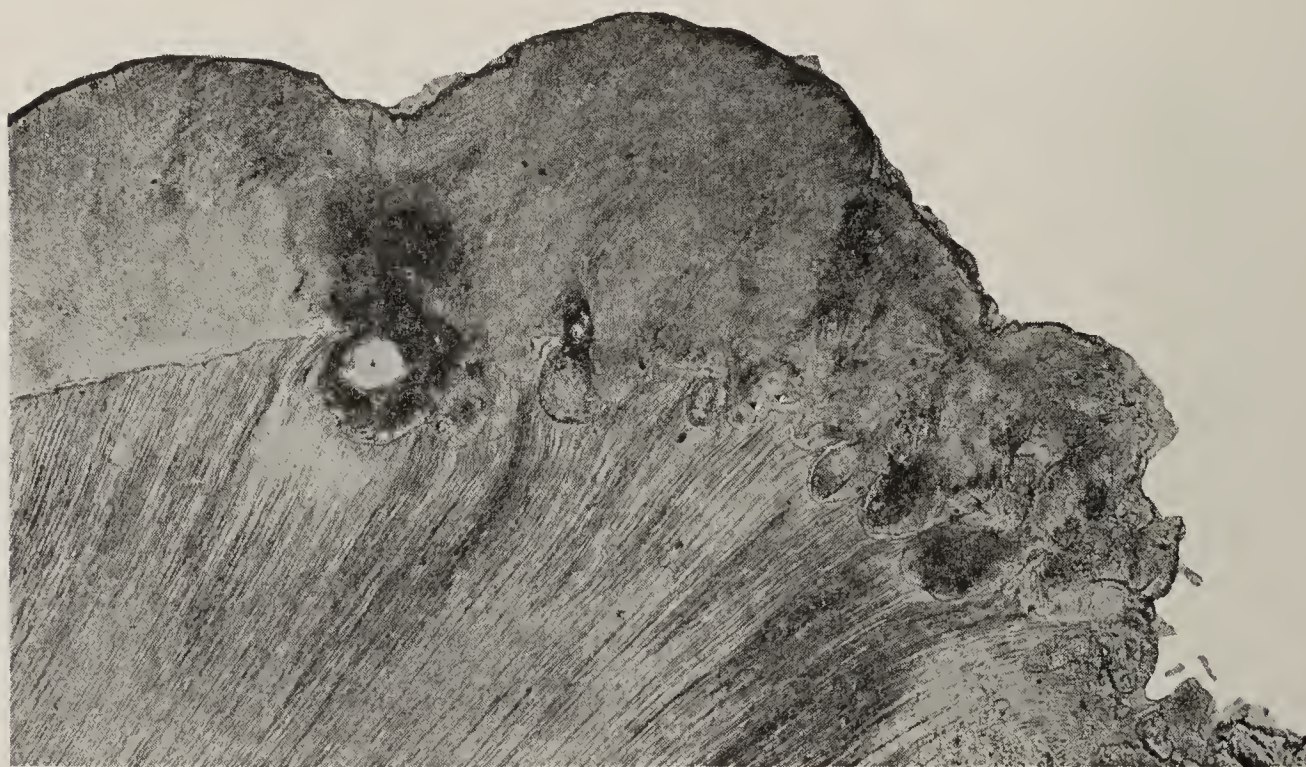


FIG. 104. Portion of enamel from near the gingival line on the buccal surface of a second molar from Doctor Callow's case.

deformity of, the enamel had no relation to contemporaneous lines of calcification. Histologically, although there were scattered interglobular spaces, there were no markings in the dentin that bore any relation to those that occur in hypoplasia. Either of these were sufficient to distinguish it as something different. In all of the teeth, from incisors to third molars, the deformity was greatest on the axial surfaces and least on the cutting edges and cusps. The surfaces were extremely rough and uneven, presenting sharp spiculæ or knobs and deep pits in the utmost irregularity of form. Over some of the cusps the enamel seemed to be normally thick, but did not have the smooth glazed surface of normal enamel. Only occasionally a small area would show the normal smoothness. In most of the teeth the enamel assumed a normal appearance suddenly near the cemental line, and this normal part generally encircled the tooth, joining the cementum in a normal line.

Figures 104 and 105 are photomicrographs showing the peculiar histological characteristics of the enamel. In most of its parts the dento-enamel junction is lost in a wild jumble of circular whorls or protrusions of enamel into the dentin. Quite a number of these whorls are hollow and empty, while some are filled with amorphous material, but all of these, without exception, are lined with enamel, usually in the form of segments of whorls, as these are found in



FIG. 105. Buccal cusp of a second bicuspid, from Doctor Callow's case.

the bottom of other enamel pits. In some this lining is very thin. Some of these hollows communicate with the surface by very small tubelike openings, while others seem to be closed on all sides. In occasional patches, even where the enamel began in these whorls along the dento-enamel junction, the rods to either side straight-

ened up and closed over them into a fairly well formed enamel. Still, most of the formed enamel is a wild, twisting, curving and bundling of enamel rods. With all of this the enamel formed seemed to be of normal hardness in every part. In a considerable number of places the enamel is plunged deeply into the dentin in long prolongations that were too large and long to permit photographing with any lens with sufficient amplification to enable the structure to be distinguished. The illustrations show the characters of the



FIG. 106. White spot in the enamel of an otherwise normal tooth. In the white area the enamel rods have no cementing substance between them. They have a covering on the surface, however, that has the usual hard glaze common to the surface of normal enamel. This is Naysmith's membrane.

departure from normal very much better than it can be portrayed in words. There was no indication of any other abnormal condition of the patient.

Many of the teeth were badly decayed and it is represented that all of them, whether decayed or not, were abnormally sensitive to sweets, heat and cold, and to acid fruits. Also, that this condition of sensitiveness had been persistent since the eruption of the teeth. This sensitiveness was so continuous and severe that

it led finally to the removal of all of the teeth for relief. This unique case stands alone. The definiteness of the deformity and the perfectness with which this definite histological character was repeated in each tooth examined, indicates that it is a deformity to which the teeth are liable. This is emphasized by the frequent observation of the tendency seen along the line of the dento-enamel junction to form scallops and whorls in teeth otherwise normal. Nothing is known of the conditions leading to this kind of deformity.

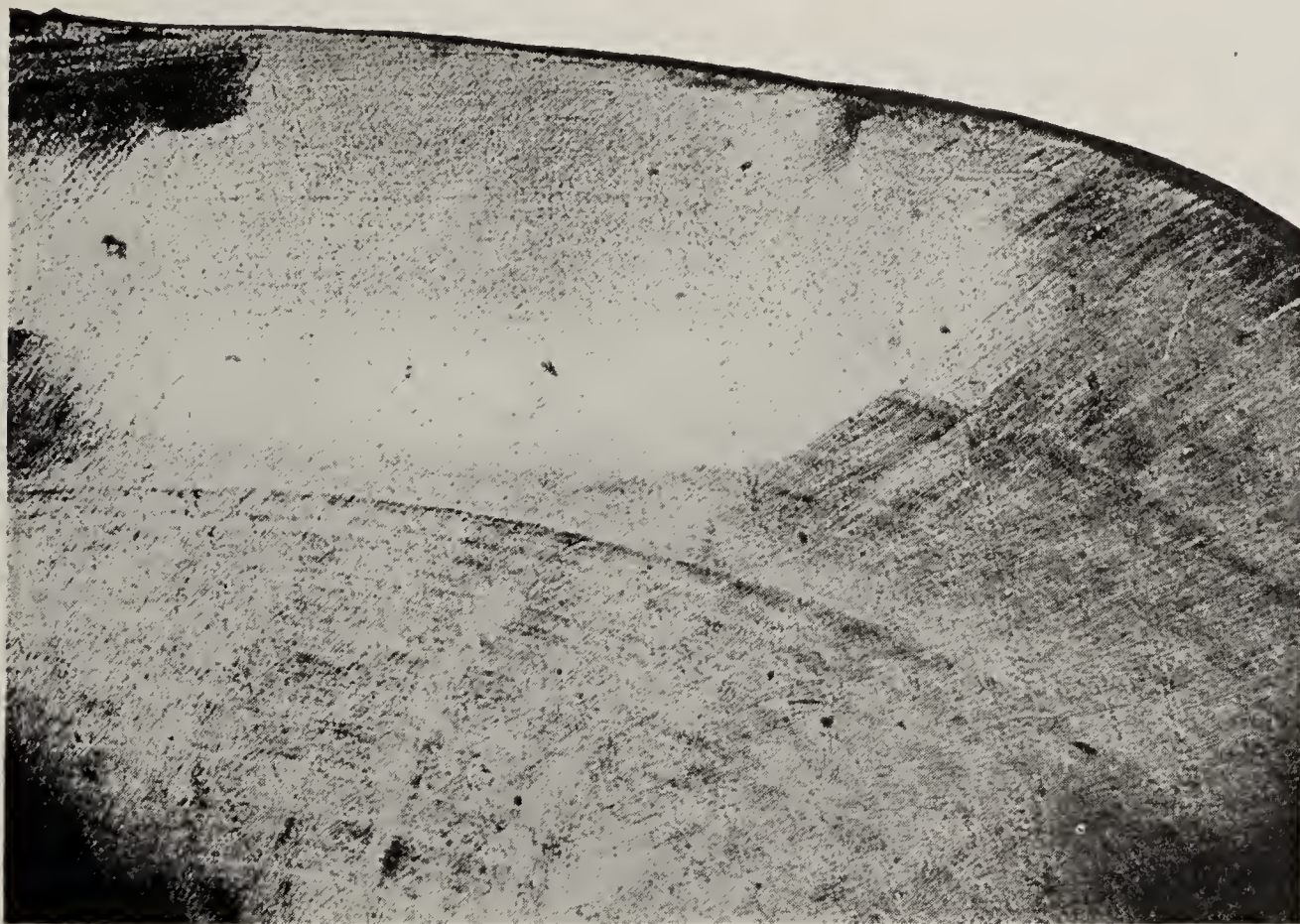


FIG. 107. White spot in enamel. At the right, little bundles of rods are seen to be without cementing substance between them.

WHITE SPOTS IN THE ENAMEL.

White enamel is seen in occasional white or ashy gray spots occurring in the enamel of teeth otherwise normal in color and form. These white spots are usually small and are covered with the ordinary glazed surface of the enamel, so that an exploring tine will glide over them the same as over the perfect enamel. If, however, the spots are large, this glazed surface fails to cover the central portion, being projected but part way from the margin toward the center. In that case, the central area is rough, and an exploring tine enters the enamel without difficulty. Such spots as these latter are rare. Sometimes such a spot shows discoloration about its central portion or radiating through it in irregular lines or blotches. The smaller white spots covered with the glazed membrane are common in any great school clinic where large numbers of persons are present for dental operations.

A histological examination of these shows the enamel rods to be normal in their formation and continuous with the rods deeper in the enamel, which is altogether normal in form and color. Generally the smaller white spots that appear on the surface of the enamel do not extend through its thickness. It often ends abruptly in a line following the incremental lines of enamel formation, i. e., the lines of Retzius, as seen in Figures 106 and 107. In the area of the white spot there is no cementing substance between the enamel rods. This is the histological characteristic of all of these white spots examined. This is, therefore, a dystrophy affecting the formation of the cementing substance between the enamel rods the same as certain of the pits in the enamel are a dystrophy of the enamel rods.

WHITE ENAMEL.

Fourteen teeth were received from Dr. D. J. McMillen, of Kansas City, which had been extracted by Dr. John Prunty, of Boyd, Texas, for one patient, all of which were deformed in what, from macroscopic examination, seemed a similar manner to that described in Dr. Callow's case. The teeth were very dirty with blood stains and from being handled, which obscured some of their most notable characters, but a closer examination showed the enamel to be soft. It could easily be picked to pieces, and evidently much of it had been lost in this way since the teeth were extracted. The axial surfaces were made up of irregularly formed spiculæ that rendered them extremely rough. Many of these had been broken, so much so, indeed, that it was with some difficulty sections were made showing the condition at the time the teeth were extracted.

The enamel was white through its entire thickness, not the white bluish color of enamel, but the white color of unglazed white paper. The enamel had seemed so frail that the teeth were soaked in thin balsam and then thoroughly dried, in order to retain the spiculæ of enamel.

This case proved to be something entirely different from Dr. Callow's case, histologically. The dento-enamel junction was perfectly normal in all its parts. Next to the dento-enamel junction the enamel was in perfect form. But after a slight growth the enamel rods broke up into bundles that became smaller, and evidently these bundles had ended in spiculæ. None of these were found that had not been broken, although there were patches with the mucoid film formed in the mouth still over them after grinding the sections, which showed conclusively that the spiculæ had not been broken after the teeth were extracted. Figure 108 shows this and indicates very well the manner of the formation of these spiculæ. Evidently the finest of these had been broken after the extraction of the teeth. In many places very little enamel remained.

This enamel throughout all its parts was almost wholly without the cementing substance between the rods. Figure 109. Histologically, this was the principal deformity. The rods themselves were doubtless fully hard, but they were not cemented together and broke apart with the greatest ease. Indeed, much of the enamel came to pieces after it was mounted and the rods became scattered in the balsam. There is no explanation of the formation of the spiculæ which constituted the principal outward deformity. In the mouth the teeth must have had a dead paper-white appearance.

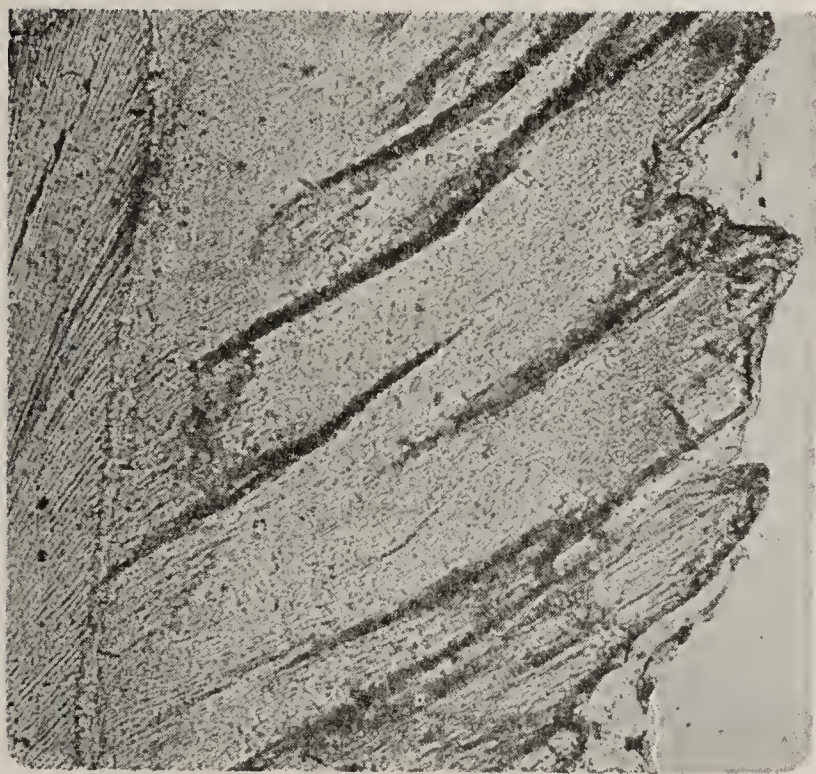


FIG. 108.

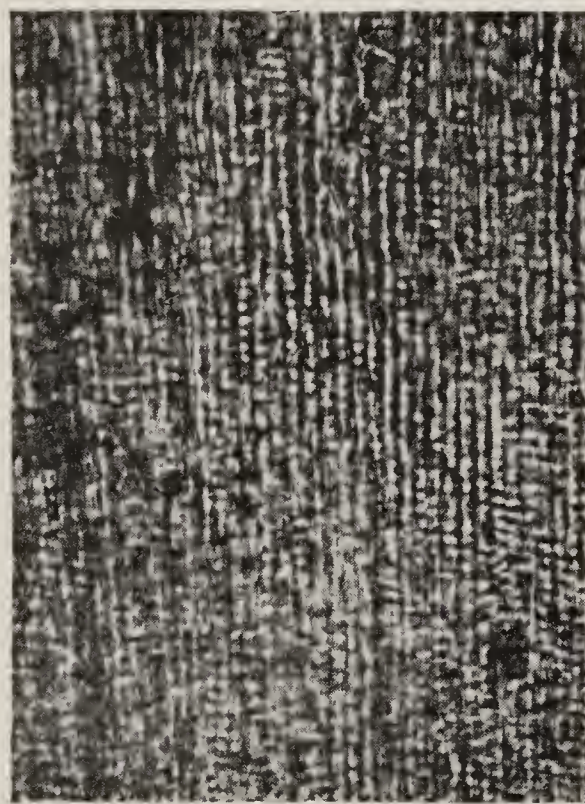


FIG. 109.

FIG. 108. Enamel from near the cusps of a tooth in Doctor Prunty's case, showing the enamel rods breaking into bundles which end in spiculæ. This enamel had no cementing substance between the enamel rods. Its color was a dead paper-white.

FIG. 109. A scrap from one of the best parts of enamel found in Doctor Prunty's case, showing the rods to be without cementing substance between them.

But one other case has been seen which presented a general absence of the cementing substance between the enamel rods. A laboring man came into the clinic at Northwestern University Dental School, whose teeth presented this dead paper-white appearance. Every tooth, and every part of every tooth, was absolutely white. There was no deformity as to form, although Nasmyth's membrane, which usually covers the enamel and forms the glaze of the surface, was absent. The teeth were of usual size, of good contour, and regular in the arch. He said they had always been so and he had been greatly annoyed because of the attention their peculiar color attracted. The man was twenty-eight years old. There were some points on the cusps where the enamel was worn enough to show the dentin, but generally the wear was not excessive. He said he could chew food as well as anybody. There were

three small proximal cavities in the bicuspid; otherwise the teeth were sound.

One cavity was partially excavated; the dentin was apparently of usual firmness, but the enamel seemed to crumble to pieces easily. Not only the walls of the cavity crumbled, but a sharp explorer could be easily pushed into the enamel of other teeth anywhere. Some of the cuttings from the enamel walls of the cavity well beyond the decayed area were distributed in glycerin under a cover-glass, and with the microscope well-formed enamel rods were seen that looked much like those that had been separated by a very weak acid, or those taken from the whitened enamel in backward decays.

This condition of the enamel had not rendered the teeth more than ordinarily liable to caries, as was shown by the general soundness of the teeth.

This condition resembled the white spots so often seen in the enamel of teeth that are in the main perfectly formed; and is undoubtedly of the same character, Figures 106, 107. The only difference seemed to be that the usual white spots are covered with a very perfect glaze, or Nasmyth's membrane, so that a sharp instrument will glide over them. This man's teeth had no such glazed surface. A sharp explorer would catch anywhere with very little pressure. In fact, it would not glide over the surface at all. The teeth evidently had not a normal Nasmyth's membrane. The enamel in the two cases was about the same as tested with cutting instruments.

One other, somewhat similar, case has come under observation in which the incisal portion of the incisors and cuspids and the occlusal portion of the bicuspid and molars were covered with normal enamel, but a large part of the axial surfaces were white enamel, much of which lacked the glazed covering, or Nasmyth's membrane. At all points this glazed membrane was projected to some distance from the normal over the abnormal enamel.

These cases, taken together with the frequent occurrence of white spots, led to the supposition that the failure of the cementing substance between the enamel rods is a special form of dystrophy or abnormality in formation to which the enamel is liable. The occurrence of this in isolated spots, which are usually of an ashy white color, is not very uncommon, but its occurrence in the whole of the enamel in the teeth of a person is certainly extremely rare. Only the two cases mentioned have been observed, one with abnormal form, the other with normal form.

Nothing seems to be known of the pathology that brings about this condition.

The study of such cases is of great importance, as it may lead to further knowledge of the formation of this tissue. Certainly the facts developed show that either the functioning tissue or the

functioning of the tissue that forms the enamel rods is so different from that which forms the cementing substance between the rods that the rods may be formed and the cementing substance fail. Also, we have seen in the illustrations many failures of the enamel rods with the space filled in part with something else apparently without histological form. This something may be the cementing substance.

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EROSION OF THE TEETH

ILLUSTRATIONS: FIGURES 111-127.

EROSION is a term applied to a peculiar and very characteristic loss of substance of the teeth, beginning in the enamel, or upon its outer surface, and slowly working its way inward and spreading, destroying and removing the substance of the tooth as it progresses. At first it presents no symptoms except this loss of substance, and this looks like a facet that would be left after grinding slightly with a very fine stone. There is no softening whatever, but simply a wasting of the substance, leaving a perfectly smooth, polished surface; a surface so smooth and polished and hard that an explorer passed over it will glide just as smoothly upon the eroded surface as upon the enamel that is perfect. The facet first formed gradually deepens and widens, progressing very slowly in most cases, until the enamel has been cut through. Then the dentin wastes away in a similar fashion, and so smoothly that there is no line of demarcation between the enamel and the dentin. When the enamel has been penetrated and the dentin begins to be eroded, the dentin becomes very sensitive. This sensitiveness is characteristic of erosion if in teeth with vital pulps. Teeth that have lost their pulps may suffer from erosion in precisely the same way, except that in these there will be no sensitiveness.

Erosion is usually slow in its progress. The facets may appear upon the enamel and be seen for a considerable time before the enamel is penetrated — a year, two years or more. It proceeds directly and steadily, in a small proportion of cases, until the teeth are destroyed, requiring from three to ten years, or even more, to cut through the crown of a tooth. In many cases the progress ceases spontaneously, or the progress may be intermittent.

The position in which erosion occurs is most commonly the buccal or labial surfaces of the teeth. It is uncertain whether it appears oftenest upon the incisors or upon the bicuspid and molars. Sometimes, however, it will begin upon the proximal surfaces, and a few cases have been observed upon the lingual surfaces. When it has begun upon a surface of a tooth, the general rule is that it does not involve other surfaces, i. e., if it has begun upon a labial surface there is rarely a disposition to begin upon a proximal surface or a lingual surface, but it will be confined to labial and buccal surfaces; it will, however, involve other labial surfaces in the neighborhood. For instance, if it begins upon a labial surface of a central incisor — which is not an uncommon place of beginning — it is likely to involve the labial surface of the other central incisor and the labial surfaces of the lateral incisors and cuspids. If it

begins upon a first molar it is liable to involve the second and third molars and the bicuspid, and in most of these cases it will be bilateral, though occasionally it is unilateral, not occurring upon the opposite side at all. It may also involve the upper teeth after having affected the lower teeth, or vice versa, but continuing upon the labial or buccal surfaces, not involving other surfaces of the teeth. If it begins on proximal surfaces, proximal surfaces only will be affected. But it will involve tooth after tooth.

DIAGNOSIS OF EROSION.

One who has carefully studied the conditions of the eroded areas in a few cases should have little difficulty in recognizing it in any of its forms. It should be remembered that the form or location of the eroded area is not distinctive, for the reason that a great variety of forms of area and of location are presented. The area of eroded surface is always smooth and glossy, and a sharp explorer held lightly in the fingers glides over it the same as over enamel. This distinguishes erosion from beginning caries, but does not distinguish it from abrasion. All facets occurring on the occlusal surfaces of the teeth should be regarded as abrasion. An examination should be made that will certainly exclude abrasion from any cause such as the rubbing of the particular part against another tooth, or rubbing by some artificial appliance, or the possible grinding by a stone for any purpose. With these excluded, a definite facet of any form that is hard, smooth and glossy, is distinctive of erosion. So long as this is in the enamel only, it will be the only symptom. A tooth with a vital pulp will usually become sensitive when the dentin is reached, but in cases that have made much progress slowly in dentin, the pulps of the teeth may become much calcified, and the sensitiveness will disappear on account of the cutting off of the dentinal fibrils by the calcification. Obviously there will be no sensitiveness when erosion occurs in pulpless teeth. In case the progress of erosion ceases, the sensitiveness of the exposed dentin soon disappears also, and the dentin may become discolored. All of these points must be considered in making a diagnosis of erosion. It will be seen from the foregoing that the one fact of loss of substance without apparent mechanical cause, leaving a smooth, glossy surface in dentin, or in enamel, or in both, is the distinguishing feature of erosion.

FREQUENCY OF EROSION.

As to frequency of occurrence, erosion is rare as compared with caries. No reliable statistics are available as to the percentage of persons who have erosion of their teeth. It seems probable that the number is larger than most observers would estimate. Apparently the incidence of erosion is increasing. Doubtless many cases are overlooked by practitioners. The evidence on these points, however, remains very uncertain.

Among the adult patients applying at the clinic at Northwestern University Dental School, about one per cent of erosion is found. These people are mostly friends of the students. They are not of the very poor nor of the wealthy. Erosion of every variety of form is found, but the rounded cuts across the teeth that tend to become stationary, or cease to progress, seem to be in the majority. Many of these cases stop spontaneously before any considerable injury is done.

Erosion is much more frequent in some certain classes of people than in others. Considerable inquiry has been made among practitioners regarding this. Some seldom see a case, while others find it very frequently; especially those whose practice is confined closely to very well-to-do people find it most frequently and of a character to do the greatest injury. It is very frequent among well-to-do Jewish people. One dentist reported erosion of the teeth of a young man of twenty, his father, his grandfather and his great-grandfather, all of whom were examined during the same week; all were Jews. This practitioner was convinced by his observations that erosion is hereditary. They are practically all descendants of a few Jewish families who settled in this country many years ago, who have been very much devoted to the maintenance of their especial set. From the descriptions first given of erosion among these people, it was expected that much of it would be of one character as to form, but there was a wide variety of form, so much so that one could not say that any one particular form prevailed to the exclusion of others. There were two cases of marked erosion of proximal surfaces, cutting between the bicuspid.

FORMS OF EROSION.

DISH-SHAPED AREAS. One of the very common forms of erosion may be described as a dish-shaped excavation, in which the center of the eroded area is deepest, and from this it rounds up to the surface of the enamel in every direction. This may attack a central incisor first and involve the teeth at either side of the tooth first attacked, hardly ever exactly bilateral, but usually more extended on one side of the mouth than the other, destroying the labial surfaces. Its place of beginning varies from the middle of the gingival third to the mid-length of the crown. Mesio-distally it is usually about the center, but if there are irregularities in the positions of the teeth it is most likely to begin on the most prominent part.

It first forms a little facet upon the enamel, then destroys more and more, and finally passes through to the dentin, cutting without any distinction whatever between the dentin and enamel, and increasing the size of these facets until the whole labial surface has been removed, not touching the proximal surface, and not touching the incisal except as it is approached from the labial. This form is represented in Figure 111. There is frequently an offshoot

from the true dish shape toward the incisal, particularly in the central incisors and cuspids above and below, and sometimes in other teeth.

Figure 112 also presents the dish shape of the eroded areas, but in this case it has two complications. There is recession of the gums and the dish-shaped portion of the erosion is largely in the cementum, but cutting the cementum, enamel and dentin without distinction. This complication of recession of the gums with erosion is not uncommon. The erosion never passes under the margin of the gum, and while the gum may be tumified or swollen, it is

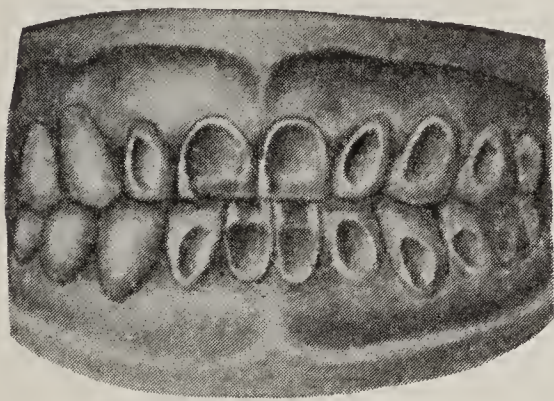


FIG. 111.



FIG. 112.

FIG. 111. A case of erosion occurring in the teeth of a woman twenty-eight years of age. These are dish-shaped areas, with projections toward the incisal edge, which are plainly seen in the lower central incisors. Otherwise it is a case of typical dish-shaped areas. A picture by the author from the same cast was published in the "American System of Dentistry."

FIG. 112. The dish-shaped areas complicated with recession of the gum, with projections extending from the dish-shaped areas toward the incisal edges, in the upper teeth. These extensions have a very characteristic squareness of their angles. Patient, a man forty years of age.

rare that it ever laps over into the eroded area, differing markedly from caries in this respect. Some of the cases look as though the recession of the gum may have been caused by the erosion, but many cases are seen of recession of the gum of similar character without erosion. Also many cases are seen of erosion cutting away the teeth very close to the gum without recession of the gum. It would therefore seem that there is no causal relation between the two. Figure 112 also has a second complication not so frequently seen, in the squared-out projections towards the incisal from the dish-shaped portion which were noted in Figure 111.

WEDGE-SHAPED AREAS. These are the "*keilförmige Defecte*" of German writers. This form usually has its place of beginning near the margin of the gum, and as seen in its beginning looks like a little groove cut across the crown of the tooth from mesial to distal. These gradually deepen, and soon the case looks as if a wedge-shaped piece had been cut out of the labial surface of the tooth, presenting a flat side reaching toward the gingival and a square shoulder toward the occlusal, as if filed away with a square file. In many of the cases the cut is as square and the angles as sharp as they could be made with such an instrument. In others

there is more inclination to rounding of the angle in the deeper part of the cut. See Figure 113.

A number of cases of this form have been observed in which the teeth were cut through so deeply that they finally broke away, cutting through the calcified pulp without any distinction whatever from other parts of the dentin. These wedge-shaped areas occur both in the upper and lower jaws, less frequently in the incisors in the upper jaw, but more frequently in the bicuspid and molars. It is not very uncommon to see this form in the bicuspid and molars, and some irregular forms or dish-shaped areas in the incisors and cuspids.

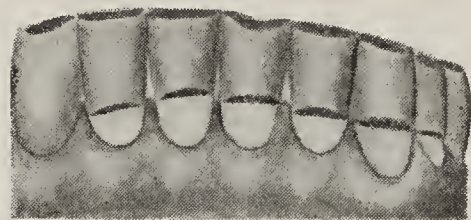


FIG. 113.



FIG. 114.

FIG. 113. The wedge-shaped areas of erosion of the "keilformige defecte" of German authors. An excellent example of that form of erosion in which a wedge-shaped piece is removed. In this case the thin edge of the wedge is toward the gingival margin. Only the lower teeth are eroded. Patient, a woman about forty years of age.

FIG. 114. A modification of the wedge-shaped areas of erosion in which the bottom of the cut is well rounded instead of a sharp angle. This form is frequent in the bicuspid and first molars. Patient, a man thirty-five years of age. No other erosion had occurred in the mouth.

In Figure 114 is shown what appears to be a modification of the wedge-shaped areas of erosion, occurring in two bicuspid. In the deeper parts the cuts are well rounded from the occlusal toward the gingival margins, but are flat mesio-distally. The form is that of a segment of a cylinder.

Figure 115A is a specimen of wedge-shaped cutting, occurring in the incisors of a young lady about nineteen years old. Three casts of this were made about one year apart, watching its progress. The picture presented here is from the first cast taken, and the erosion at that time had been noticed only about one year. It has not gone deeper since, but has spread considerably more toward the incisal.

Figures 115B and C are two views of an upper central incisor,* which illustrates unusually deep wedge-shaped cutting. It has cut deeply into the dentin, including secondary dentin which had been built to close the pulp chamber, and the perfect smoothness of the eroded surface is continuous, as an instrument is passed over the enamel, original dentin and secondary dentin. In this case the other upper incisors and the cuspids were similarly involved.

FLATTENED AREAS. The tendency seems evidently toward the flat form, which is prominently presented in Figure 116. This case was from a man about sixty years old, a laborer. He had apparently taken no care of his teeth, and stated that he never

*Specimen presented by Dr. Harold G. Ray.

used a brush in his life. This case presents very remarkable peculiarities in the triangular patches of enamel remaining on the upper right central and lateral, and the lengthwise cutting of the lower left lateral. The gums were in very good condition.

Figure 117 presents another case which, while it looks similar, is really quite different. This occurred in a man about sixty-five years old, and had been watched for a number of years by a dentist

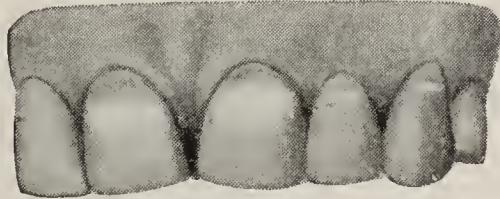


FIG. 115A. An early stage of what appears to be a modification of the wedge-shaped erosion with a tendency toward the flat form. The extension is toward the incisal and there is an absence of the cupping from mesial to distal. In these, the cutting is generally shallow. Patient, a girl nineteen years of age.

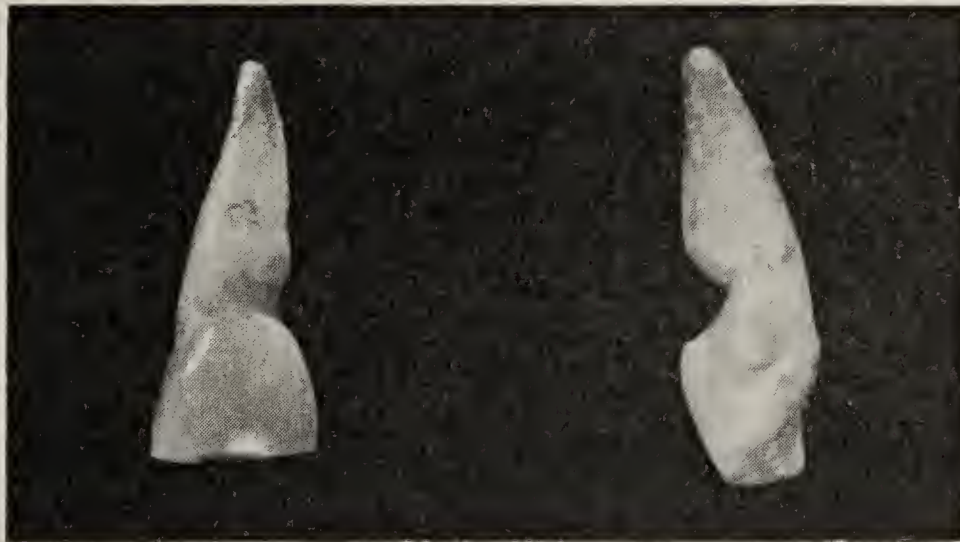


FIG. 115B.

FIG. 115C.

FIG. 115B, 115C. Two views of an upper central incisor with a very deep wedge shaped erosion. All of the upper incisors of this patient were in the same condition.

noted for his accuracy of observation. The course of the erosion was very rapid, occupying but between three and four years. In this case it began very near the center of the labial surfaces and spread more rapidly toward the incisal than toward the gingival, and the incisal edge was soon invaded. The proximal surfaces also began to lose material and the centrals spread apart. There was not the perfect smoothness of surface usually seen in cases of erosion, and the teeth became so very sensitive that artificial crowns were placed upon them.

The next case, Figure 118, is somewhat similar in its nature. This case, as viewed from a distance of a few feet, gave the impression that the upper incisors were retained temporary teeth, but closer inspection dispelled this idea at once. The gentleman was about twenty-eight years old, and the normal wear where the lower teeth had occluded against the lingual surfaces of the upper incisors was unmistakable, showing that it had not been very long in

progress. The teeth were so sensitive that the patient was greatly annoyed, and was seeking relief. The teeth were cut in such a way as to cup them out slightly upon the labial surfaces. The proximal surfaces were wasting perhaps as much as the labial surfaces, but were cut rather squarely, almost as though they had been filed. The teeth showed no traces of scratches of any kind, but they were not as smooth and glossy as most cases of erosion; they were more like the case last described.

IRREGULAR AREAS. In Figure 119 is presented another class of case entirely which will be described under the head of irregular areas. In this case there are grooves passing across the teeth from mesial to distal, not always at the same angle nor of the



FIG. 116. The flat form in an advanced stage, but with a cuspid but little advanced, and a triangular patch of enamel still left on one lateral and one central incisor of the upper jaw. The incisors are cut rather deeply at the gingival portion of the crown. The gums are in good condition. The lower left cuspid presents a perfectly flattened labial surface from mesial to distal, but it is a little concave from incisal to gingival. The lower left lateral is cut through its length as though it had been cut in a planing mill, it is so level and perfect, with almost perfectly squared edges on either side of the cut. A man about sixty years of age; a laborer, who avers that he never owned or used a tooth brush.



FIG. 117.



FIG. 118.

FIG. 117. A flat form which ran a very rapid course, complicated with erosion of the proximal surfaces. The small figure on the right is a labio-lingual section, intended to give more definite information as to the loss of substance. The incisors were destroyed within five years after the erosion was first seen. From a man of wealth about sixty years of age.

FIG. 118. A flat form complicated by erosion of the proximal surfaces, which are also flat. No erosion of the lower teeth or any of the bicuspids or molars. The upper incisors had overlapped the lower normally before the erosion shortened the teeth. This case presented extreme sensitiveness of the eroded areas. A man twenty-eight years of age, a bookkeeper.

same depth, some appearing upon the mesial most, and others appearing upon the distal most, and so on. Occasionally the groove may extend not more than half across the labial surface of the tooth, but cutting very deeply; in fact, they may present almost any form, but generally in grooves from mesial to distal.

In one case a single groove was cut across the teeth at about the middle of the length of the crown, less than two millimeters

wide, and cut squarely in, fully one-half through the tooth, affecting the two centrals and one lateral. In this case it was cut so squarely that it seemed reasonable to place restorations with the expectation that the progress of the cutting would be stopped. The restorations remained, with no cutting about the margins until the time of the gentleman's death, some ten years later. In a large collection of casts, a good many of these irregular cuts may be found.



FIG. 119.

FIG. 119. A type of the irregular form. Some of these present singular extravagances. Man, about forty years of age.



FIG. 120.

FIG. 120. The pattern form, or figure cutting. In this particular case, in the right central incisor one horizontal and one perpendicular groove are cut, which are joined, forming a right angle, and there is also a groove in the labial portion of the incisal edge joining at a right angle with the perpendicular groove. A similar figure is being outlined upon the opposite central incisor. Patient, a girl, nineteen years of age.

FIGURED AREAS. In Figure 120 is presented an illustration of what may be designated as figure cutting in erosion, that is, cases in which the erosion takes a form of some particular complex type. In this, the central incisor on the left of the figure, a rounded groove is cut across the labial face of the tooth near the junction of the middle and gingival thirds. From that a groove is cut along the mesial border straight to the incisal edge, and then the incisal portion is squarely grooved across the labial portion of its edge. The cutting upon the fellow central incisor had started in the same way and was following the same course. In a similar case the groove reaching the incisal edge extended to the lingual along the mesial margin of the lingual surface. The teeth were long cusp teeth. They were practically unworn and there was no reason whatever to attribute the grooving on the lingual surface to abrasion.

Figures 121, 122 and 123 illustrate another case of figure cutting that is much more extensive. In this case there was no grooving of the incisal edge, or along the incisal edge, only a slight broadening of the groove on the mesial side, and it will be noted that a similar groove was being cut upon the distal portion of the labial surface, while there is a curious half dish-shaped form on the distal end of the groove running mesio-distally. In this cutting, the angles with the surface of the tooth are very sharp, but the bottoms of the grooves are well rounded. The depth

of the cut from the transverse groove to the incisal is shown in Figure 121, while the depth of the cut across the labial surface is shown in Figure 123.

It was clearly apparent in examination of the tooth, that the groove running mesio-distally had cut through the calcified pulp. The incisal edge of the tooth had been much worn by ordinary abrasion. The V-shaped cut in the root of the tooth was made with a file for the examination of the pulp canal.

Many gold foil restorations have been made with a view of arresting erosion, and generally the erosion progressed beside the restoration as though none had been placed. The margins of the



FIG. 121.



FIG. 122.



FIG. 123.

FIGS. 121, 122, 123. Pattern form more advanced. The cut across the root of the tooth was made with the file for the examination of the condition of the pulp chamber as to calcification. Notice that a second groove was starting on the distal portion of the crown. Figure 121 shows the depth of the perpendicular groove at the incisal edge, and Figure 123 the depth of the horizontal groove.

restorations stood up sharp and definite, showing no rounding. Dr. W. D. Miller, in his articles on erosion, claimed to have seen restorations of various sorts cut away by this process. A case which is presented in Figure 124 shows a gold crown, which had been placed over the first bicuspid and which seems to have been cut through. The patient was a dentist, and he claims this to be the third gold crown that had been cut through at the same point. It was placed over the tooth because it had become quite deeply eroded and was extremely sensitive. The second bicuspid below is protected by a gold crown, placed for a similar reason. It has shown no signs of injury. It will be noted that erosion is in progress in other teeth in the neighborhood.

In Figure 125 is presented an illustration of a case of erosion of proximal surfaces. The picture is reproduced from pencil sketches made during the progress of the case. The progress of this case was observed over a period of about seven years, nothing

being done or attempted in the way of treatment. The picture represents the case as it was first presented. It appeared as if holes had been bored between the teeth, cutting about equally from either tooth. These grew larger and larger and the interproximal gum tissue receded slightly until the teeth were cut away so much that they broke, leaving the roots in position. During all of this time, the margins of the cut at their junction with the enamel surface were sharp, presenting no observable rounding. This has been observed in the molars and bicuspid in several cases, and usually the teeth have been extremely sensitive. In one case all of the molars had been removed from the upper jaw because of pain in chewing food and in efforts to clean the teeth.

A very rare case of proximal surface erosion is shown in a reproduction of a radiograph in Figure 126. There is a very narrow wedge shaped cut entirely across the distal surface of the upper



FIG. 124.

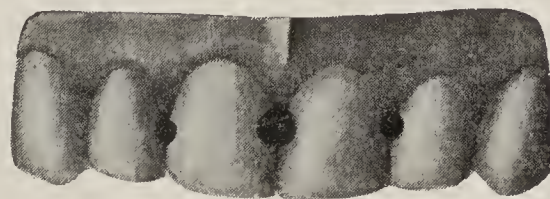


FIG. 125.

FIG. 124. Dish shapes. The lower bicuspid and first molar are gold shell crowns, which were placed because of the extreme sensitiveness of the eroded areas. The crown on the first bicuspid is the third shell crown which has been worn through by the erosion. The white cement may be seen through the hole in the crown. Man, forty years of age; a dentist.

FIG. 125. Proximal surface erosion. A case of a young man, about nineteen years of age. When first observed the teeth were in the condition presented in this illustration, showing round holes through between the teeth. These enlarged slowly, and the teeth broke away about seven years later.

first molar. At the time the radiograph was taken the cut was about 1 mm. in width occluso-gingivally and about 3 mm. deep. An explorer could be passed from buccal to lingual with the point in the depth of the groove, which had the characteristic smoothness of erosion. There is also a cut in the distal surface of the second bicuspid, which is not clearly shown in the illustration.

Figure 127 illustrates a case of extensive lingual erosion of the incisor teeth. A little more than the incisal half of the enamel has been entirely removed from the lingual surface of both lower central incisors, the left lateral incisor, and the cuspid. This occurred within a two year period in a man about fifty-five years of age. There is a large mesial inlay in the cuspid and it was necessary to trim the margins more than half a millimeter to make them even with the eroded surface.

The selection of cases for illustration has been confined to a few which seem to represent the more typical varieties of form. Variations from these, however, are constantly coming up, almost every new case presenting characters peculiar to itself. Many of them, even when the teeth are badly cut and deformed, are so clean and white that the deformity is scarcely noticeable a few feet away. But occasionally cases are seen that give a very bad appearance.



FIG. 126.

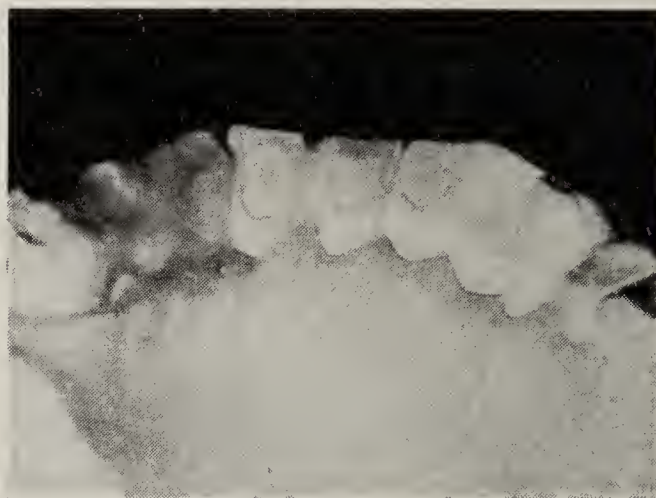


FIG. 127.

FIG. 126. Case of wedge shaped erosion on the distal surface of a molar.

FIG. 127. A very unusual case of lingual erosion, involving the lower central incisors, left lateral incisor and cuspid. All of the enamel has been cut away from about three-fourths of the lingual surface and from the incisal edge. The mesial restoration in the cuspid, which was of even contour with the surface of the enamel, showed a prominence of more than half a millimeter.

ETIOLOGY OF EROSION.

The cause of erosion is involved in the utmost obscurity. Most observers seem to have drawn their opinions of its causation from the observation of the circumstances attending a comparatively small number of cases coming under their personal observation. As the cases differ very materially in form of affected areas and surroundings, these views differ with the groups of cases observed. These opinions may be found in the literature reaching back some two hundred years. Perhaps a better and more condensed idea of this may be given by grouping these opinions into classes, following, in some degree, the order in which they have been prominent in the literature. They may be thus summarized:

(1.) Erosion is a result of faults in the formation of the tissues during the growth of the teeth; conditions in after life have little or nothing to do with it.

(2.) Erosion is caused by friction, most generally of the tooth brush carrying some abrasive tooth powder.

(3.) Erosion is the result of the action of an acid in some way, as yet unknown to us.

(4.) Erosion is the direct result of the action of a secretion of certain diseased glands in the mucous membranes that lie

directly upon the areas being eroded. This secretion is generally claimed to be acid in its reaction.

(5.) Erosion is a process of absorption similar to that of the removal of the roots of deciduous teeth or the occasional absorption of the roots of the permanent teeth. The absorbing tissue is the diseased portion of mucous membrane found lying in the eroded areas.

(6.) Erosion is in some way the action of an acid, the development of which is associated with the gouty diathesis.

(7.) Erosion is effected by alkaline fluids acting upon the basic substance and setting the calcium salts free, which waste away with a polished surface.

(8.) While dental caries is the result of the action of an acid developed by microorganisms, erosion is effected by the enzyme of the same or similar microorganisms.

(9.) Erosion is caused by disordered pulp due to an abnormal occlusion.

(10.) Erosion may be caused by an exudate from the gingival crevice due to traumatic occlusion.

In discussing briefly the principal points in these views, some will be found overlapping more or less with each other, or separated only by the introduction of certain factors supposed important by one observer, but not considered so by another.

THE FIRST SUPPOSITION mentioned seems to have been the older view. It was clearly expressed by John Hunter in 1778, by Fox in 1806, by Bell in 1825, and by a number of others following these, and finally by Garretson as late as 1890.

Hunter supposes that erosion is due to some certain imperfection in the formation of the tissues of the tooth, because of which the substance gradually wastes away, continually leaving a smooth surface. His supposition seems to have been that conditions in after life had nothing especially to do with the condition.

Fox and Bell do not differ materially from this view, but Bell adds the supposition that in the case of the dish-shaped areas of erosion, the tissues of the tooth have been deposited in whorls or such forms as favored this kind of wasting. Today such views seem very strange; but it should be remembered that when these men wrote nothing was known of the histology of the teeth, and their view did no violence to facts known in their time. It is now known that such faults in the formation of the tissues do not occur as the basis of these cases.

THE SECOND SUPPOSITION, i. e., that erosion is caused by friction, and generally by the tooth brush loaded with abrasive powders, was held by John Tomes and many others in England, America and Germany, and is still held by many observers. According to this view erosion is no disease at all, but is purely a mechanical injury. Conditions present in quite a number of

the cases seen, if grouped together and considered alone, would give strong support to this view. These cases nearly all belong to the more indefinite shapes of the wedge-like forms, and the cases which occur in the cementum after, or with, recession of the gum. A number of persons who hold this view describe and illustrate these forms only. This has led to the suspicion that in some regions, particular forms are more frequently met than others. It seems certain that in the central part of the United States one would not observe many cases of the wedge-shaped areas without finding varieties of form that would be very difficult to explain as resulting from abrasion by the tooth brush. When Charles Tomes edited another edition of his father's work, he had seen cases that he believed could not have been made with the tooth brush, and he rewrote the chapter entire. Dr. C. R. E. Koch (*Dental Cosmos*, Volume 15, 1873, page 463) tried by every device he could think of to produce the conditions seen in erosion by the use of brushes and brush wheels, aided by acids in some cases and by alkalies in others. His conclusion was that it could not be done in any of these ways. He did not have the modern electric motor to run his brushes, however.

Dr. W. D. Miller, of Berlin, Germany, published (*Dental Cosmos* of January and February, 1907) the results of two years' work on the etiology of erosion. He announced his belief that it is caused by weak acids or gritty tooth powders, or by both, assisted by the tooth brush. He seemed to be convinced that the tooth brush is the main factor, and that what we have designated as erosion is nothing more nor less than abrasion brought about by these agencies.

Dr. Miller's laboratory experiments were performed in the following manner while he was engaged in these studies. Having placed a number of teeth in wax, gutta percha, or similar substance, somewhat in the form they would be in the mouth, he saturated a cloth with a weak acid solution (different solutions being used in various experiments) and laid it lightly over the teeth so that it would touch only on the most prominent part of their crowns. After this had remained in a moist chamber intended to prevent too much evaporation, for a predetermined length of time, the cloth was removed and the preparation placed on a machine run by an electric motor, and the teeth were vigorously brushed for a given number of minutes or hours, as determined for the particular case. The cloth was then resaturated with the acid solution, placed again upon the teeth, and the preparation returned to the moist chamber for the predetermined number of hours for that experiment. This was kept up day after day for weeks or for months, the experiments being much varied as to acid solutions, time, and brushing; also, certain experiments were being carried on without the acid solutions, and others by the use of tooth powders in use by the people. In some of the experi-

ments the brushing was done by hand, as by this method more variety of motion could be used.

In these ways he had certainly produced results that looked very much like many cases of erosion as seen in the mouth, especially those of the partially dish-shaped, the wedge-shaped, and the flattened varieties. None of his artificial productions, however, had the sharp, clean-cut margins so often present in the real thing as it occurs in the mouth. Yet it is probable that if these specimens had been presented as erosions that had occurred in the mouth in the ordinary way, their genuineness would not have been questioned. Certainly Dr. Miller demonstrated the possibility and the probability that teeth are often injured by vigorous brushing with gritty powders, persisted in several times per day for many years together. His evidence is not conclusive, however, that all erosions are produced in this way, even admitting that they may be assisted in part by an acid that may be present in the fluids of the mouth.

One thing particularly Dr. Miller seems to have ignored, and that is the extreme sensitiveness that is so often present in cases of erosion. It is known, however, that Dr. Miller had not finished his investigation of the subject at the time of his death, and that the peculiar sensitiveness occurring in erosion would have been considered by him later if he had had the opportunity. This is regarded as peculiar to erosion, admitting, however, that cases occur in which there is no history of this symptom. The examination of many cases justifies the belief that frequently sensitiveness is soon annulled by the rapid calcification of the pulps of the teeth. This has the effect of cutting off the connection of the dentinal fibrils of the crown of the tooth with the remaining pulp tissue. Then sensitiveness is ended. Therefore, many of the bad cases of erosion have not been sensitive for several years. This calcification has been a result of the condition of irritation communicated through the dentinal fibrils.

During the time of this irritable condition there is generally a history of the cessation of brushing, for the reason that the friction of the brush can not be borne. In the case illustrated in Figure 118, the young man said he had used a brush before his teeth became so sensitive, but most of the loss of substance had occurred afterward. This is but a repetition of the history obtained from many persons.

THE THIRD SUPPOSITION — that it is the action of an acid — has been held by many persons, and in one form or other probably has more adherents today than any other. How it is that an acid can so act to cut away the substance of the tooth, leaving a hard, polished surface which is a constant characteristic of erosion, while in laboratory experiments and in caries as it occurs in the mouth the effect is a gradual softening by the solution of the calcium salts, is left unexplained, unless Dr. Miller's work noted above may be so regarded. As yet no acid has been found that will remove the

whole of the tissue, calcium salts and basic substance, without previous softening.

Some experiments were made by the author on a different plan (reported in the "American System of Dentistry," Volume I, page 1003). Having noticed in some experimental work on the metals that the action of very dilute acids was different in the still condition as compared with the action in currents, this was tried upon teeth. Here also a difference was found. In a rapid current of a solution of one part of hydrochloric acid to four hundred parts of water, maintained for five days continuously, teeth were cut away in forms quite similar to erosion, the cut surfaces remaining hard and smooth, while other portions of the teeth were not softened. The loss of substance occurred only where the current broke around the teeth in a certain way. While this experiment is impossible of comparison with anything that can occur in the human mouth, it demonstrates the possibility that the action of acid solutions may be modified in some degree by conditions under which they are placed. Thus far, however, no modification has been discovered that will in any degree account for effects like those seen in erosion under conditions that seem possible in the human mouth.

More recently, Bernard B. Bedanes (*Dental Cosmos*, May, 1930, p. 477) has suggested that erosion is caused by the presence of soluble oxalates in the saliva. He claims to have procured specimens of uncombined oxalates from two erosion patients at the bedside in the morning before the patients had lifted their heads. This precaution was necessary to prevent the oxalates from combining with the calcium in the saliva. He states that the oxalates result from a disordered protein metabolism. Dr. Bedanes also claims to have produced erosion artificially on an extracted tooth by the use of a pellet of cotton saturated with acid potassium oxalate.

THE FOURTH SUPPOSITION — that erosion is caused by the secretion of certain glands in the mucous membrane of the lips and cheeks, that these glands become inflamed, or hypertrophied, from some unexplained cause, and emit an abnormal secretion which acts upon the teeth in this peculiar manner. It is certainly true in many of the dish-shaped eroded areas in incisors particularly, that a certain part of the mucous membrane is found to be raised in a form that fits into the excavations in the teeth.

Many of these cases have been examined and the facts found as stated. It has also been found in these cases that the *prints of teeth not eroded were clearly outlined in the mucous membranes*. After a careful and somewhat protracted study of these phenomena, the conclusion is that the little swellings of the mucous membranes are caused by the erosion, rather than the erosion by a secretion which they emit. In a considerable number of persons the prints of the teeth — the upper incisors particularly, and some-

times the bicuspid and molars also — will be found in the lips or buccal membranes, and these are formed in the same manner.

This explanation is strongly emphasized by the fact that erosions occur in cases in which the mucous membranes never touch the teeth, as in the somewhat rare cases of erosion of proximal surfaces, and in one case observed in a girl sixteen years old, in which the bicuspid were deeply eroded in dish-shaped forms, where the tissues of the cheek had been destroyed before the teeth came through the gums, so that no mucous membrane could have touched them.

THE FIFTH SUPPOSITION has certain points of similarity with the fourth in that the same raised points of the mucous membrane are described as fitting into the eroded areas. But instead of the erosion being caused by an acid, the supposition is that this tissue acts as an absorbing organ, and that the result is really an absorption similar to that by which the roots of the teeth are cut away. Several writers speak of having seen the usual lacunae of absorbed areas in roots of teeth and in areas of bone undergoing absorption, in these eroded areas. This supposition necessarily carries with it the idea that certain actively functioning cells become fixed against the tooth tissue and keep that position long enough or steadily enough to effect this result. The same objection to this theory applies as to the previous one, namely, that cases of erosion occur in positions in which no mucous membrane, or other tissue, is in contact with the areas being eroded.

THE SIXTH SUPPOSITION is that it is caused by an acid that is developed in association with the gouty diathesis. Perhaps the most notable article upon this supposition is that by Dr. Darby, of Philadelphia (*Dental Cosmos*, Volume 34, 1892, page 629). Dr. E. C. Kirk, of the same city, has expressed somewhat similar views. On this point there is much negative observation. Yet it may be expected that the cause of erosion will eventually be found to depend upon some change in the body fluids giving oral secretions favoring these results; but how, by what, and how localized, are the questions. For the present it seems that there are no certain data in support of the theory connecting it with the gouty diathesis.

THE SEVENTH SUPPOSITION — that erosion is caused by alkaline fluids acting upon the basic substance of the tooth, setting the calcium salts free, which waste away leaving a polished surface — is one of the more recent. It has an individuality of its own. The supposition that an alkaline condition is maintained in these localities for any considerable part of the time is certainly contrary to much the larger number of recorded observations in the literature. Further, in Dr. Koch's experimental work mentioned above, he found that he could not dissolve either enamel or dentin in alkalies until the calcium salts had been removed, or partially

removed by acids. The enamel particularly contains so little organic matter that it would seem impossible — first, that its organic matter could be removed in that way, and second, that if it could be accomplished it would not effect the disintegration of the tissue. The dentin has a much larger proportion of organic matter, but direct experiment seems to show that it has sufficient calcium salts to protect its organic matter from solution by alkalies. Therefore, until it can be shown experimentally that in some possible form alkalies will act to disintegrate these tissues, this supposition must be set aside.

THE EIGHTH SUPPOSITION is set forth by Preiswerk in his "Zahnheilkunde" ("Operative Dentistry"), 1903, page 200. It is the supposition that erosion is caused by the same or similar microorganisms as those which cause caries, but by the action of their enzyme, not by the action of their acid products. It is well known that a number of the microorganisms of the mouth which form acids in the presence of the carbohydrates, such as sugar or starch, will grow well in nutrient material devoid of these substances, but in that case will form no acid products. Preiswerk contends that the human saliva is normally alkaline, and that it is a mistake to suppose that the enzyme of these microorganisms is necessarily a peptonoid substance, as these act in an acid medium. He claims to have found evidence that in alkaline or neutral conditions their enzyme is a trypsin similar to the trypsin of the pancreas which acts in the presence of an alkaline reaction. Under these conditions this enzyme acts upon the basic substance of the dental tissues, dissolving the basic substance, setting the calcium salts free, which are washed away during the chewing of food, the motions of the lips, fluids of the mouth, and in the artificial cleaning of the teeth. He claims to have in some degree proven this proposition by experiment with trypsin derived from the pancreas, which he found to act upon the basic substance of the teeth.

It seems quite possible that trypsin might act upon bone in this manner, possibly upon dentin, but that it should so act upon enamel, which has no more than three per cent of basic substance, seems out of the question. Erosion always begins in the enamel, except in those cases in which it begins in the cementum after the recession of the gum tissue, and practically always cuts the enamel as smoothly with the dentin as if the two were one and the same tissue.

In the absorption of the roots of the temporary teeth, and not very rarely of the permanent teeth as well, all of the parts, basic substance and calcium salts, are cut away as one tissue. In the absorption of bone by the normal physiological process, the same thing occurs, and in studies of these processes it has often been observed that the noncalcified bone corpuscles suffered the same fate. But even this process balks at enamel. Enamel has often

been in position to be acted upon by absorption, but this has failed. It has often been observed that almost the last trace of dentin had been removed from the crowns of deciduous teeth by physiological absorption, but never any part of the enamel. It appears at the present time that these facts place this process out of the question, even if the far more general observation — that the condition is acid in erosion — should be shown to be an error.

THE NINTH SUPPOSITION — that erosion is caused by a disordered pulp due to an abnormal occlusion, does not account for the localization of erosion or for the fact that erosion affects teeth not containing vital pulps.

THE TENTH SUPPOSITION — that erosion is caused by an acid exudate from the gingival crevice due to traumatic occlusion, is being given some credence. Charles F. Bödecker (*Dental Cosmos*, 1933, page 1056) reports that by placing small triangular slips of litmus paper firmly against the teeth and gingivae, he demonstrated the presence of acid where erosion was present, but where there was no erosion, no acid was found. In considering this supposition it should be emphasized that acid acting on enamel always results in a softening of that tissue, a condition never observed in erosion.

In 1935, examination of the saliva of twelve patients made in the chemical laboratory of Northwestern University Dental School disclosed the interesting fact that although in every case the saliva contained the normal content of calcium and phosphorous, in no case was the saliva supersaturated with the above elements, which is invariably true in normal patients. In most cases the saliva was slightly under the saturation point but in two or three patients was just to the saturation point. If the above be true in all cases of erosion, it would explain the slow dissolution of the enamel without softening but would still leave unexplained the localization of erosion and the various forms so frequently observed. Apparently this warrants further study.

Finally, there appears to be no theory that has not features that seem to render it impossible. Therefore it is necessary to leave the subject in this very unsatisfactory condition, hoping that an early solution of the difficulty may be discovered. It seems highly probable that this will be found connected with some systemic dyscrasia, but if so, the conditions leading to its strict localization will require explanation.

TREATMENT OF EROSION.

At present no treatment with the view of cure or of stopping the progress of erosion, is known, that gives promise of success in any considerable variety of the cases. There should be strict inquiry as to the patient's habits of cleaning the teeth in every case, particularly with respect to the use of abrasive tooth powders

and pastes. A sufficient use of clear water and the brush should do no harm. Dr. Miller's work in the production of erosion seemed to indicate that he was not very successful in producing erosion with the brush and water, even when much brushing was done with the electric motor. With the powders, however, the teeth were worn away. He also found sharp grit in many of the tooth powders in use. The brush used with water regularly and sufficiently, but with moderation, will certainly keep the teeth in good condition as to cleanliness. Those who have connected erosion with the gouty diathesis have made some effort for the relief of the general condition with the hope that the progress of the erosion might be controlled, but thus far no very considerable benefits have been reported.

Considerable effort has been made to reduce the evil results by inserting restorations with few successes, and some of the apparent successes were doubtless due to a coincident spontaneous stoppage of progress of the erosion, rather than from the influence of the restorations. In the case illustrated in Figure 111 the eroded areas were restored with gold, removing all that remained of the labial and buccal surfaces. This was effective for the teeth so treated, and fortunately the erosion did not spread to other teeth. The case was observed over a period of about fifteen years. This was done before the modern process of porcelain crowns had become successful. The treatment was very objectionable from the esthetic standpoint, but it gave the patient the full use of the teeth with perfect comfort.

Restorations have generally been of no other value except to limit sensitiveness and depth of cutting, unless all of the surface being eroded was removed, except possibly in some of the narrow cross cuttings and a few of the wedge-shaped areas. The erosion will go on beside the restorations and continue spreading, leaving the margins of the restorations standing as they were placed.

One who has had a wide observation of erosion and of its progress from year to year may do much good by making restorations in certain selected cases. These should be for those deep cuts across the teeth that show little or no disposition to lateral spreading. In a number of cases of this character the groove has been cut fully to the mesial and distal surfaces, and restorations have been placed without other preparation. These have generally been successful. If the restorations are made without the extension mentioned, the erosion will soon make the extensions and new restorations will be required. The wedge-shaped forms have in a few instances done well with restorations, and certainly the depth of the cutting can be materially limited. However, the restorations have failed to stop the spreading. With the dish forms the porcelain jacket crown is the better treatment when the cutting is so deep as to cause a disfigurement. In other forms of cutting, restorations seem to be of no use except to limit the sensitiveness for a consid-

erable time, and to prevent depth. In the great majority of cases, however, it will be better not to make restorations. As a general rule, restorations should not be made in the treatment of erosion. That which now promises the best results is to keep watch of the cases, and at the proper time cut away the remaining parts of the crowns and place artificial crowns.

RELIEF FROM SENSITIVENESS. The sensitiveness of these areas may be relieved, temporarily at least, by severe burnishing. This may be done with any rather heavy instrument having round edges, such as the beaver-tail burnisher. Burnishers, in various rounded forms, see Volume II, Fig. 304, may be used in the engine. Heavy pressure should be made while the burnisher is rotating. The burnisher should be applied to each area sufficiently to give it a thorough rubbing. The first application will cause sharp pain momentarily, but afterward there will be less, or no pain. This treatment will generally relieve the sensitiveness for some time. It may be repeated when necessary.

When erosion has progressed so far that the teeth have become much disfigured, the crowns should be cut away and replaced with porcelain jacket crowns.

In watching the progress of many cases of erosion a rather large number will be found that become stationary without known cause, and so remain indefinitely. These cases will, of course, vitiate many of the supposed good results of treatment by restoration, or any form of treatment intended to limit or to stop the progress of erosion.

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ABRASION OF THE TEETH

ILLUSTRATIONS: FIGURES 131-140.

THE term "abrasion" is applied to *abnormal wear* of the teeth — more wear than occurs from the normal processes of mastication. That this statement of the case is correct, any one may become satisfied by the careful examination of some hundreds of individuals, from forty to sixty years old, and tabulating the condition of the teeth as to abrasion of their occlusal surfaces, and any other conditions discoverable that may be regarded as influencing it. The conclusion will certainly be that there is some unknown influence responsible for an abnormal amount of abrasion of the teeth of certain individuals. Yet, the friction of mastication controls the form of the abraded surfaces of the teeth.



FIG. 131. Abrasion of the temporary teeth of a child who was five and a half years old.

There are great differences among persons as to the amount of wear of the teeth. Among the large majority there is a certain degree of abrasion of the teeth that is apparent in nearly all persons forty years of age or more. This is not considerable; and is insufficient to cause disfiguration or to require attention. In a comparatively few individuals there is much greater wear; so much that persons are occasionally seen, the crowns of whose teeth are worn to the level of the gums at the age of forty. A greater number will be met with, whose teeth are worn so much as to disfigure the mouth and the features also, by the closing of the lower jaw too far, interfering with the position of the lips and shortening the chin. Indeed, some persons appear almost as those who have lost their teeth, though there may be a considerable part of the crowns remaining.

Abrasion of the temporary teeth is not uncommon. In an occasional case the crowns will be much worn. The author recalls

one case in which all of the teeth of the temporary set were worn practically to the level of the gums. A case of moderate abrasion of the incisor teeth of a child five and a half years of age is shown in Figure 131. When this child's mouth was closed, the upper incisors fully overlapped the lowers so they could not be seen and the lower incisors touched the gingivae to the lingual of the upper incisors.

In abrasions that progress rapidly, there are certain features which show that the use of the teeth in mastication has much to

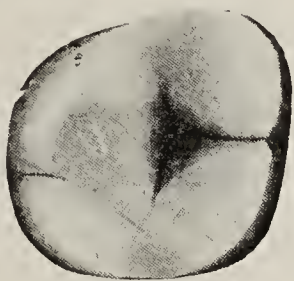


FIG. 132.



FIG. 133.

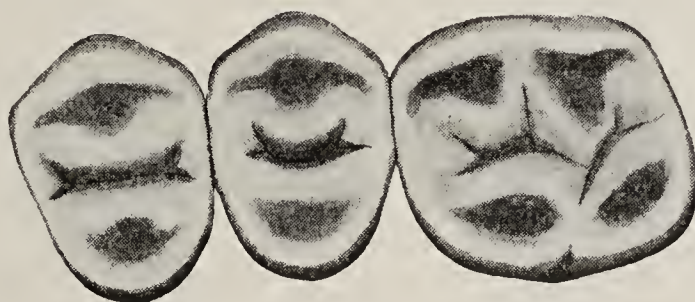


FIG. 134.



FIG. 135.

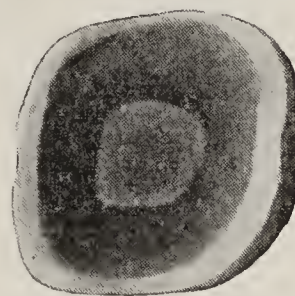


FIG. 136.

FIGS. 132, 133, 135 and 136 show progressive abrasion of a molar tooth which has worn until the calcification filling the pulp chamber is exposed.

FIG. 134. The bicuspids and first molar, the cusps of which are worn through the enamel and the dentin is cupped out.

do with the form of the worn surfaces. Usually in the beginning, whether the amount of the abrasion is eventually to be great or small, the cusps (or some certain cusps) are worn flat, and the developmental grooves, which were prominent features before, gradually disappear and the occlusal surface generally becomes flattened, as is seen in the molar tooth in Figure 132. When the abrasion has gone further, the dentin, under the prominence of some or all of the cusps, becomes exposed, as shown in Figures 133, 134. The dentin, being softer, wears more rapidly than the enamel, and cups out into rounded cavities of more or less considerable depth. The dentin becomes yellowish in cases that are rapidly wearing

away. If the wear is slower, it becomes darker, and, in many cases, almost black. In the meantime, the remaining enamel retains its white color. If a section is made through one of these darkened areas, and this is photographed by reflected light, a cloud will appear stretching to the pulp chamber following the direction of the dentinal tubules. The pulp chamber is already undergoing changes. The horns of the pulp chamber have shortened and many of the cases show that secondary dentin is being deposited on all sides of the pulp chamber. With the greater abrasion shown in Figure 135, all of these changes will be markedly increased. Up to this time the patient is apt to have periods of sensitiveness of some of the abraded surfaces and more especially when a certain cusp first shows an exposure of the dentin. This sensitiveness comes and goes for a time and then is likely to disappear entirely and permanently. With the continuance of the abrasion, the condition appears as shown in Figure 136, in which the entire occlusal surface proper has disappeared. The enamel remains higher than

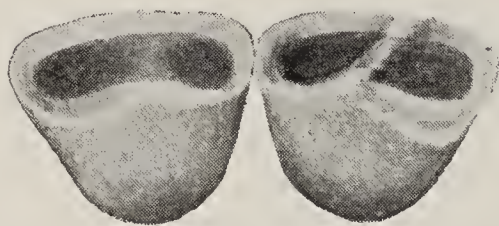


FIG. 137.

FIG. 137. The upper central incisors, showing extensive abrasion.



FIG. 138.

FIG. 138. A labial view of the teeth. The lighter area indicates the probable form of the portion removed by abrasion.

the dentin. The dentin is cupped out in its center, sometimes very deeply, and in its center the form of the pulp chamber shows as a lighter area. The bulb of the pulp has become completely calcified. A section of this calcified area will show that true dentin was formed for a time after the pulp chamber began to diminish in size. After this has continued for a space, more or less of the dentinal tubules disappear, and in a little space farther all have disappeared, and the rest of the mass is a clear calcification showing none of the histological forms of dentin. In some cases the calcification will be incomplete; one side, angle or other part not having closed. This latter is more often seen in the incisors.

Figures 137 and 138 illustrate a case of extensive abrasion of the upper central incisors. The movement of the mandible is indicated by the ridge on the upper left central, corresponding to the space between the incisal edges, or surfaces, of two lower incisors. In Figure 138, the probable extent of the abrasion of these teeth, beyond what would be the normal wear, is illustrated.

Figure 139 shows a badly worn lateral incisor, split mesio-distally, and photographed by reflected light. It will be seen that most of the pulp is solidly calcified and that there is no sharp line showing the original outline of the pulp chamber. But the light streak running down centrally in what was once a large pulp chamber is a minute opening which contained a filament of living pulp tissue when the tooth was extracted. This had ceased, however, to have any connection with the dentinal tubules of the crown of the tooth, and all sensation of the dentin in that part of the tooth had been lost.

Another case, a central incisor split mesio-distally and photographed by reflected light, is shown in Figure 140. The outlines of the pulp in the crown of the tooth are distinct. In studying a ground section of this, it was found that all dentinal tubules



FIG. 139.



FIG. 140.

FIG. 139. A central incisor that has been badly worn. The pulp is all calcified except a mere shred that shows as a white line.

FIG. 140. A central incisor, the greater part of the crown of which is worn away. The pulp is completely calcified far into the root.

had disappeared almost at once on the border of the whitish area outlining the pulp chamber as it must have been when the calcification was only a little advanced. From that time all connection between the dentinal tubules in the tooth crown and the pulp of the tooth had been broken. The dentin of the crown of the tooth was as completely insensitive as if the pulp had been removed. This is the usual history of the calcification of the pulps of teeth in abrasions. In incisors worn down to the gum line, the form of the pulp will usually be seen as a clear spot in the center of the worn area.

If there is a tooth in the mouth, among these badly worn teeth, that has lost its occlusion by reason of one or more teeth of the opposite jaw having been extracted early in life, it will not be worn down. An examination of its pulp chamber, however, will show the same changes as found in the pulp chambers of those that have

been worn. Further, in the examination of the pulp chambers of the molars, and following the lines of contemporaneous calcification, which may generally be quite accurately made out, it will be found that the additions to the floor and axial walls of the pulp chamber have been quite as great as the additions between the pulp chamber and the worn surface. In other words, this calcification, while certainly stimulated by an impression made upon the nervous system by the wear on the surface of the exposed dentin, has been brought about by some influence through the trophic nerves, and has had its influence upon the pulps of all of the teeth, including those that happened to escape abrasion, as well as those that were actually worn away. Cases occur, however, though they are comparatively few, in which abrasion exposes the pulp of the tooth; sometimes one or two of the many that are worn away in one person's mouth. Sometimes this occurs in an individual cuspid, or an incisor, or the lingual horn of an upper molar; in each case probably the individual horn of the pulp was unusually long and was caught early in the wear.

With the abrasion and loss of vitality in the tooth crown, there is also a loss of strength of the dentin, and the attachment of the enamel to the dentin becomes enfeebled. This fact is important in undertaking restorations in these teeth. The same thing occurs in teeth in which the pulp has been long dead. In either case, restorations need to be more securely anchored than in normal dentin.

When teeth become badly worn, the enamel margin around the worn area often has worn much less than the dentin. This enamel is then liable to split off under the force of mastication and become ragged and rough. Sometimes this will cut and irritate the lips, cheeks or tongue; and it should be ground smooth and polished.

Often the abrasion is very uneven on the different parts of the teeth. Frequently the occlusion is such that the wear comes mostly on the buccal cusps of the lower teeth and the lingual cusps of the upper teeth. Normally the upper incisors overlap the lower and the result at first is wear from the labial of the lower and the lingual of the upper incisors. If the wear of the molars allows the jaws to close more, or shortens the teeth generally, the occlusion of the front teeth tends to come directly end to end, by the movement of the lower jaw forward as it closes more. At the same time, it often happens that in the molars and bicuspid the abrasion becomes more and more one-sided on the individual teeth. The lower molars and bicuspid are worn on the buccal sides, while the upper are worn on their lingual sides. This will go on until the wear of the occlusal surfaces is such that the teeth slide together instead of meeting with each other squarely in occlusion.

The treatment of abrasion is given in the chapter on the technical treatment of the teeth during the adult period. See Volume III, Figures 1116 to 1128, with the accompanying text.

CARIES OF THE TEETH

CARIES, or decay, of the teeth consists of a chemical dissolution of calcium salts by lactic acid. It always begins on the surface of the tooth and destroys first the enamel, then the dentin, and, if neglected, causes the death of the pulp.

Caries is the most widespread of all diseases, affecting the teeth of persons of all races and of all ages. Only a few primitive tribes are free from caries. There is, however, an appreciable percentage of persons of all races who are completely immune to caries throughout their entire lives. Others exhibit all degrees of susceptibility.

All animals living under their normal conditions are free from caries. It is a disease peculiar to man.

In many families there is an evident hereditary disposition to dental caries. Numerous practitioners have reported members of families who exhibited much the same degree and the same age periods of susceptibility through four generations.

Caries is primarily a disease of childhood and early adult life. Neglect during the childhood period usually inaugurates a chain of events which affect the patient's health and limit his activity throughout life to a greater or lesser degree. Among these, impairment of the function of mastication handicaps the digestive apparatus and thus reduces the building and resisting power of the body mechanism.

Caries attacks and destroys the temporary teeth in the same manner and in about the same percentage of cases as the teeth of the permanent set. Failure to conserve the temporary teeth usually increases the susceptibility of the individual and contributes to the early loss of permanent teeth, particularly the first permanent molars, which are the most important of all teeth.

The early loss of the temporary molars, or of one or more of the first permanent molars, almost certainly causes irregularity of alignment of the permanent teeth, which increases susceptibility and reduces masticating power.

There are many variations in susceptibility in most individuals. On the average, susceptibility becomes much less as life advances beyond twenty-five. It is often increased in conditions of malnutrition; during adolescence; as a result of constitutional or febrile diseases, mental disturbance or periods of severe strain and many other illnesses; also in women during pregnancy. There is often marked increase in susceptibility in persons who change

place of residence, particularly from one country to another, coupled with a change in diet and living conditions.

Cavities of decay occur in fairly regular progression in the mouths of persons of average susceptibility. The pits and fissures — faults in the enamel — are likely to decay within a year or so after the teeth erupt; in the teens occur the proximal surface decays in both the posterior and the anterior teeth; finally the gingival thirds of labial and buccal surfaces may decay. There are the widest conceivable variations from this progression in many individuals.

If a pulp dies and is not promptly removed, it will become infected and involve the periapical tissues, which in turn may, for many years, be a source of infections elsewhere.

Indirectly caries induces infection of the supporting structures of the teeth, through irritation by sharp edges of cavities, by causing lodgements of food which decompose and as a result of the extraction of hopelessly decayed teeth. The extraction of a tooth usually results in the movement of others with the establishment of spaces or abnormal relations between the teeth, which permit lodgements of food about them. Thus the peridental structures become the seat of chronic foci of infection.

Extensions from either of these dental sources may involve neighboring structures of the face and neck in serious acute infections; or metastatic areas may develop, which are occasionally acute, but usually chronic. The latter may involve joints, muscles, heart, stomach, gall bladder, appendix, etc. The secondary infections are generally slowly debilitating, reducing the vigour of the individual in later years — a handicap which often contributes to an earlier demise than might otherwise occur.

Thus, dental caries, which begins as an insidious lesion, without either inflammation or pain, may gradually lead to one complication after another throughout life. It may be accompanied by very severe pain within the tooth itself; may impair digestion and nutrition as a result of the loss of teeth, with disturbance of facial contours which mar the appearance and attractiveness of the individual; may result in extremely acute and occasionally fatal infections of the head, neck and chest; and may, through the establishment of chronic foci of infection cause remote lesions of important organs and structures, which handicap activities and impair health during the later years, thus contributing directly or indirectly to the termination of life itself.

HISTORICAL.

3 ILLUSTRATIONS: 151A-151C.

More or less vague writings of caries of the teeth are found in ancient literature, most of which are too uncertain in their meaning for us to gain any clear conception of the views held

of its cause. Other writings of the specialties in medicine, such as the mention of physicians for the teeth, extraction of teeth and of artificial teeth are not of interest in this connection. Close studies of the conditions surrounding the beginning and progress of dental caries have developed very slowly. The oldest known writing which attempts a rational presentation of the cause of dental caries is by an anonymous author in the German language in 1530. A photographic reproduction of a single paragraph is presented.

**Corrosio ist eine Kranckheit vnd vehel
der zen wenn sie löcherigk vnd hol werdē
welchs am meisten den backzenē geschicht
vornemichlichen so einer ist vnnnd sie nicht
von der anhangēde speise reiniget/welchs
faul wirdt/ vnd macht darnach böse sch
arffe feuchtigkeit die sie aus frist vñ erzet/
vnd ymmer all melich vberhant nymmet
bass sie auch ganz vnd gar die zen verder
bet / vnnnd darnach nicht ane schmerzen
müssen stückicht wegk faulen.**

The statement is remarkable for its close agreement in substance to the views now held, if stated without detail. The wording is very quaint and in some unessential points the meaning is uncertain.

TRANSLATION. Caries is a disease and evil of the teeth in which they become full of holes and hollow, which most often affects the back teeth; especially so when they are not cleaned of clinging particles of food which decompose, producing an acid moisture (literally, a sharp moisture) which eats them away and destroys them so that finally with much pain they rot away little by little.

In the same volume this writer gives also the oldest authentic mention of the use of gold for filling teeth. This writer mentions Mesu as an authority, a man who lived some two hundred years earlier, who is also mentioned by other writers, but so far as is now known, none of his writings have been preserved.

The French writings in dentistry are older than the English, but do not contain very clear statements of view of the pathology of caries. Generally the statements are equivalent to saying the teeth rot away or decay away, without attempt at explanation of the process.

Fauchard wrote in French in 1728 and a translation was made into German by Augustine Duddei in 1733. Second and

third editions were published in French in 1746 and 1786. In none of these is there any statement regarding the cause of dental caries that is as definite in conception as the one quoted above. This seems to be true of all of the writings of that time.

John Hunter, writing in English (1778), expresses very clearly a different view, in which he says: "The most common disease to which the teeth are exposed is such a decay as would appear to deserve the name of mortification," with which he expresses some dissatisfaction as being an incomplete explanation of the diseased process. This, with other writings, by the same author, shows that in considering the diseases of the teeth he was following closely the lines of thought of his time of what we now know as necrosis of bones.

Fox (1806) expresses a similar view, which, with slight modification, was repeated by Bell (1825), who proposed the term "dental gangrene" to take the place of the more common terms "decay" or "caries."

This seems to have been the most common view of medical men of that time, and, with slight modifications, was repeated by most writers. All of these men regarded caries of the teeth as being a result of inflammation and as beginning within the dentin instead of upon the surface of the enamel. Köcker of Philadelphia (1830) speaks of decay penetrating the enamel from within outward, saying that it "had thus formed a natural outlet for the bony abscess."

Robertson (1835) expresses a different view, which, in its main features, agrees substantially with the earlier views of the anonymous author quoted above. But Robertson is more explicit in the detail. According to this view, caries of the teeth resulted from the action of an acid generated by the decomposition of food particles or fluids, which lodged at particular points about the teeth and dissolved out the calcium salts of which the teeth are composed. These points of lodgment were shown to be the points at which caries made its beginning, as in pits and deep grooves in the occlusal surfaces, between the teeth (proximal surfaces) or about the margins of the gums.

Regnard of Paris (1838) defined caries of the teeth as "destruction of the teeth by decomposition." His contention was that this destruction took place at the very spot where the acid was formed, or where the alimentary particles lodged and decomposed.

This was called the chemical theory of caries of the teeth. A large body of dentists, both in Europe and America, gave similar expressions of view at about this time. The idea that inflammation of the dentin had any part in its causation was denied. Also the statements of Fox, Bell, and many others, that caries began within the dentin and worked its way outward, were generally denied. Instead, it was asserted that caries always began upon the surface of the tooth, or in pits, fissures, etc., that were open to the surface.

The contentions along these differences of thought were sharp and the lines closely drawn. This brought about a much closer observation and study of the nature and form of the physical injuries inflicted by dental caries, and with this, the opinion became general that caries always began on the outside of the tooth and worked its way inward, forming a cavity. During this period also, comparative anatomists and geologists were studying closely the teeth of the living animals and those remaining of extinct animals, in which prominent differences between the structure of the teeth and the bones were ascertained. Owen gave us the word "dentin," distinguishing that which had before been called tooth bone from true bone.

A knowledge of histology began to be developed. The cell theory of the construction of organic bodies, animal and vegetable, was propounded and rapidly assumed the general form in which it stands at the present time. Makers of microscope lenses rapidly improved them because of the encouragement and patronage induced by these studies. In the midst of this, John Tomes, of London, was studying the microscopic structure — the histology — of the teeth and bones, and by 1860 this was developed almost completely as it stands today. It is true that since then there has been much more exactness of method and greater accuracy of detail. But the full foundation of our knowledge of dental histology and the development of the teeth was laid by John Tomes.

It seems that Mr. Tomes began this work with the inflammatory theory of caries strongly fixed in his mind. He found, however, that inflammation could not take place in the teeth. The histological structure of the teeth was such that there was no provision for the circulation of blood in the dentin, neither was there any provision for processes of repair of injuries. Yet the dentin was a vital tissue and it was the opinion of Mr. Tomes that this vitality must be destroyed before the part could be dissolved out by an acid, thus forming a cavity. He admitted, however, that the same agent — an acid — might do both. This gave rise to the chemico-vital theory of dental caries, which was much discussed from 1840 to 1880.

Finally Dr. Magitot of Paris (English translation, 1878) published a more extensive work than had been produced on this subject, detailing much experimentation in various ways in the endeavor to determine the exact cause of the disease. His conclusions were that caries of the teeth was produced purely by chemical substances developed in the mouth or introduced with food. This work seemed, for the time being, to establish the purely chemical theory of the production of dental caries.

In the meantime, there had been many suggestions that micro-organisms might be found to play a prominent part in the production of dental caries. The first important work published on this subject was by Leber and Rottenstein (German 1867 — English

translation, 1868) in which these authors claimed to have determined the presence of these fungi in the dentinal tubules (which were much enlarged) of carious areas. Strong corroborative evidence of the correctness of their view existed in the fact that John Tomes had determined previously that the tubules in carious areas were constantly much enlarged and filled with granules, the nature of which he could not determine. This observation by Mr. Tomes had been confirmed by the observation of others and had become fixed as an essential difference between dental caries and a simple solution of the calcium salts of a tooth by an acid. Still, the work of Leber and Rottenstein made no considerable impression on the opinions held by dentists. These gentlemen wrote before the development of the staining methods by anilin dyes by which microorganisms in tissues are distinguished. Neither were they able, by means of culture methods then known, to separate microorganisms into distinct species and determine the character of each as to its power of producing fermentation or other special forms of decomposition.

Milles and Underwood of London (1881) determined definitely that the enlarged tubules in dental caries contained microorganisms, by use of the anilin dyes discovered by Dr. Koch, the German bacteriologist, but they were unable to go farther for the lack of better facilities for division of species of microorganisms and the determination of their physiological characters in the production of fermentations or putrefactions.

Dr. W. D. Miller was at work with Dr. Koch in his bacteriological laboratory when the means of cultivating microorganisms on semi-solid media was first established and was at once able to separate the microorganisms found in the mouth or in carious dentin into species, and determine the character of each in the production of acid fermentation or other forms of decomposition. The finding in the dentinal tubules of microorganisms which, when growing in artificial culture in the presence of any form of sugar or starch, uniformly produced lactic acid, which in time dissolved the calcium salts of the tooth tissue, completed the full explanation of the local changes taking place in caries of dentin, but the cause and the nature of caries of enamel was not so clearly made out.

Although the work of Miller and his predecessors may be considered to have given complete and exact knowledge of the steps in caries of dentin, many problems were left for solution, especially with reference to caries of enamel and the beginning of the process. Miller, however, pointed out that mouth organisms produce lactic acid in fermenting carbohydrates and that lactic acid decalcifies tooth enamel. He also noted that decalcification of enamel in the mouth occurs at the "retention centers;" pits and fissures, proximal surfaces, etc.

These investigations afforded a new basis for the study and analysis of the clinical behavior of cases of caries, and it was

possible for G. V. Black (1890-1910) to work out most of the details that have to do with the location of the initial lesion and map out quite exactly the extent and boundaries of both the susceptible and immune areas on the surfaces of teeth. He was also able to demonstrate that adequate cavity preparation and accurate restoration of the lost structure, based upon his clinical observations, generally prevented further extension of these carious lesions in the teeth so treated. The principle of *extension for prevention* was thus established.

The publication by G. V. Black in 1895* of a series of studies on the physical properties of the teeth, with reference to hardness and softness, as represented by the percentage of calcium salts in the dentin, called attention to the fact that the structure of the teeth was in no practical way related to the occurrence of caries, except as surface defects permitted lodgments. The findings were contrary to views generally held by members of the profession and served to emphasize more strongly the need of better understanding of the beginnings and progress of caries as a guide for its treatment. It also suggested that the causes of immunity and susceptibility to dental caries would necessarily be found in the conditions influencing the environment of the teeth.

J. Leon Williams published a series of studies (*Dental Cosmos*, 1897) of faults in the teeth of animals as compared with faults in the teeth of man. He found the structure of the human teeth much more perfect than that of the teeth of the animals. The faults in structure were less frequent in man and generally of less consequence, notwithstanding the fact that animals do not suffer from caries of the teeth, except in a few rare instances of captive animals kept in cages, some domesticated house-dogs, etc.

Dr. Williams also, 1897-1932, made extensive studies of caries of enamel in both human and animal teeth and added much to the understanding of the histologic changes that take place in enamel in decay. He was able to stain the deposits on the surfaces of early caries and demonstrate in these films the presence of microorganisms and assumed from this that the beginning of caries was brought about by fermentation of organic material in these films by the bacteria caught in the meshes of these deposits. These films he called *bacterial plaques*. See Figure 151A, a photomicrograph of a stained film of microorganisms over the surface of decaying enamel. The film moved away a little from the surface of the enamel in mounting the specimen.

Dr. Joseph P. Michaels, of Paris, presented a brochure under the title of sialo-semeiology to the International Dental Congress, Paris, 1900, which was translated into English and published in the office of the *Dental Cosmos*, 1902. He claimed to have found the means of determining definitely the existence of the conditions

* *Dental Cosmos*, Vol. 37, 1895, pp. 353, 469, 553, 637 and 737.

of susceptibility to, or immunity from, dental caries, by the examination of a few drops of an individual's saliva. Persons who had spent some time in Dr. Michaels' laboratory and looked over his work, seemed convinced that he could do so. The study consisted largely of the examination of crystals with the microscope and micropolariscope, and was necessarily qualitative rather than quantitative. Dr. Michaels died soon after the publication of his brochure.

During the period from 1906 to 1910, the Committee on Dental Science of the New York State Dental Society made a special study of the influence of the presence of sulphocyanids in the saliva. The absence of the sulphocyanid of potassium, or the presence of only a very small quantity, in the saliva, seemed to

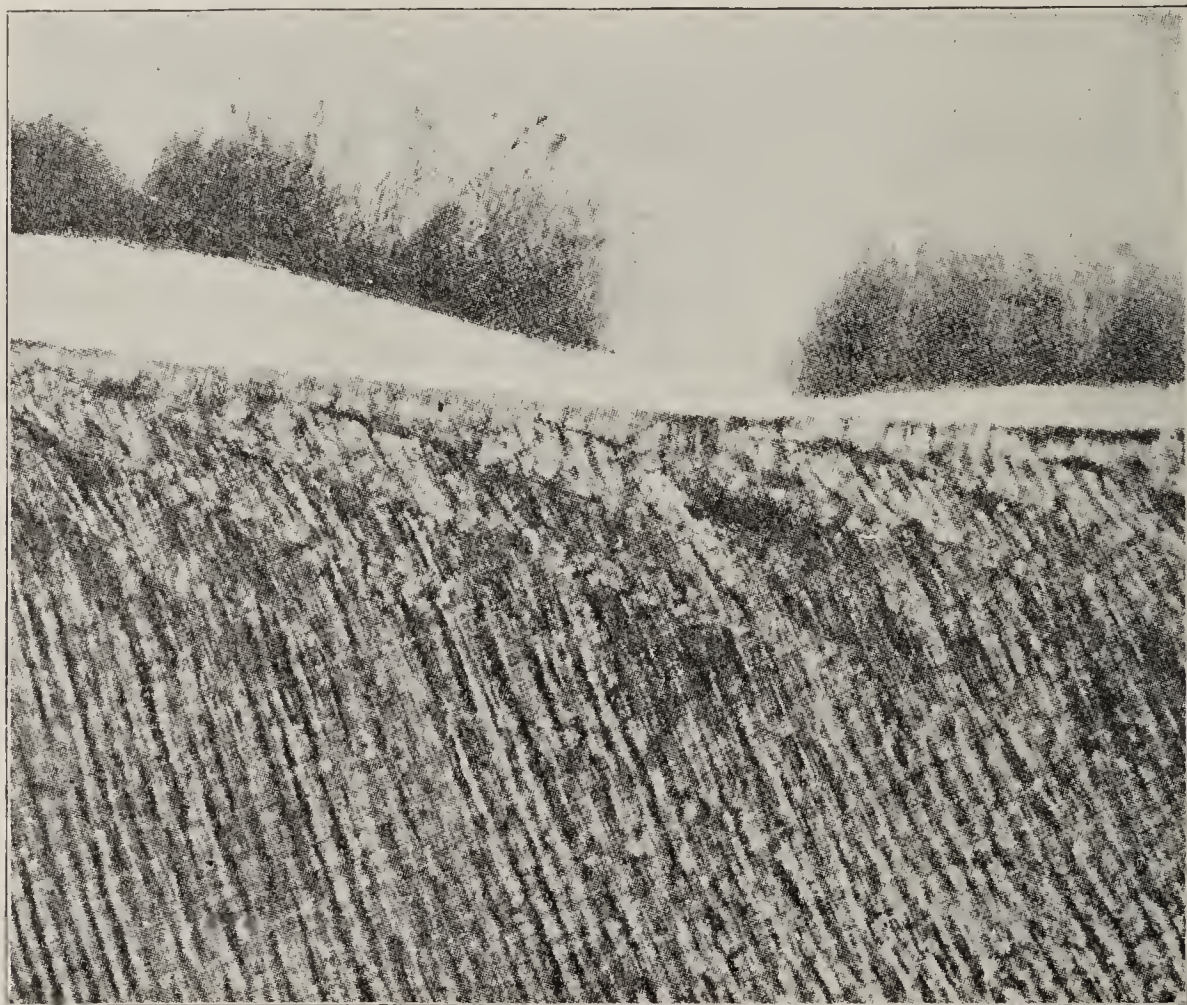


FIG. 151A. A photomicrograph showing a stained film of microorganisms over the surface of decaying enamel. In pressing down the cover-glass in mounting the specimen, the microbic film was parted slightly from the surface of the enamel. WILLIAMS.

indicate susceptibility, while the more decisive reactions, resulting from a considerable quantity of sulphocyanids, seemed to indicate immunity to dental caries. Although no very definite conclusions were reached, these studies serve to strengthen the belief that the saliva holds the secret which will solve the etiology of caries of the teeth.

While the studies of the sulphocyanids were in progress, Dr. Carl Röse, of Dresden, Germany, made an extended study of the saliva, especially with reference to its amount under different conditions of living and the variations in the salts it contained. In this work he collected an immense amount of data with reference to the

effects of the salts found in the water used for drinking and cooking purposes. In this work he developed important facts regarding the relation of these salts to the percentage of dental caries, the relation of the amount of saliva secreted by the person to the percentage of caries, the relation of salts in drinking water to the development of the salivary glands, etc. The examinations were mostly made in children, and were widely distributed in Germany, Switzerland, Denmark, Sweden, etc.

This mention of these several lines of investigation of the saliva is given here to show the direction of thought and the activity manifested, rather than for the purpose of giving specific information of its detail.

Nothing of material importance happened in the succeeding years, up to the period (1915-1920) when nutritional studies following the discovery of the vitamins disclosed the considerable harmful effects of vitamin deficiencies on developing teeth. Following this there evolved a gradual separation of investigators of the cause and nature of caries into two fundamentally different schools of thought. One of these maintains that local environmental factors are chiefly responsible for caries; the other holds that diet and defective nutrition are to be considered the primary cause. All of the details of the differences of opinion are discussed in critical papers by Enright, Friesell and Trescher (1932) and Rosebury (1933). These reviews, especially the former, have been used extensively in assembling the material presented in the preceding and following paragraphs.

The latter members of the environmental group have concerned themselves largely with bacteriologic and chemical studies of the carious process. McIntosh (1922), Okumura, Rodriguez and Bunting are in substantial agreement with respect to the importance of microorganisms of the lacto-bacillus type in producing the acid that decalcifies enamel. Bunting and his co-workers have satisfied themselves that the lacto-bacilli, which are concerned in the process, have certain typical characteristics, that these types are generally present in the mouths of persons that have active caries or are about to develop caries, that they are absent or only present as transients in the mouths of those who have no active caries or are immune, that there is some evidence for the production of immune bodies specific for these lacto-bacilli, and that caries is a specific infectious disease. Enright, Friesell and Trescher conclude on the basis of their own work and critical study of the literature that "the only microorganism commonly found in the food debris, in direct contact with progressive caries of enamel that can tolerate and produce additional acid in an environment below pH 5.0* is a lacto-bacillus having certain typical characteristics."

* The pH of a solution relates to the hydrogen-ion concentration. Distilled water is a neutral solution, of which the pH is 7. A pH of less than 7 is an acid solution; a pH greater than 7 is a base or alkaline solution; pH5 is more acid than pH6; pH9 is more alkaline than pH8.

Efforts to disclose characteristic chemical differences between the environment of carious and non-carious teeth by analysis of saliva have been fruitless, although saliva low in content of calcium phosphate has some minor significance. It was found (Vogt, also Enright, Friesell and Trescher) that very weak acid, carbonated water, even distilled water, and lactate acid citrate buffer solutions pH of 4.0 to 8.0 would etch enamel artificially. On the other hand, acids applied to teeth in saturated solutions of tricalcium phosphate did not etch teeth—that is, did not dissolve enamel—in acid concentrations of less than about pH 5.0. Since saliva is not only a saturated solution of tricalcium phosphate, but generally super-saturated, this seems to be a matter of very great importance, as only microorganisms that can produce and live in an acid environment of greater concentration than pH 5.0 can dissolve enamel in the mouth except where they may be sheltered or protected from the action of saliva.

The degradation of carbohydrates to acids has been studied and it is now known that there are five steps to this process and that the different steps require different ferments or enzymes. There is some evidence at hand to show that no one microorganism found in the mouth of patients susceptible to dental caries can supply the necessary five enzymes to bring about a rapid transformation of these carbohydrates to the final acid end product (Fosdick and Hansen, 1935). These facts and the failure of many bacteriologists (Rhoades, etc.) to develop data that harmonize the incidence of lacto-bacilli and active caries have caused Rosebury, Appleton and others to suspend judgment with reference to the significance of lacto-bacilli as the specific microorganism of dental caries, and to suggest that the question of the causative organism or organisms is still an open one.

“Within the dental field, it appears that Mellanby, Howe and McCollum have made the most extensive contributions on the relation of deficient diet and defective nutrition to the etiology of dental caries.” Mrs. Mellanby has found that the feeding of diets deficient in Vitamin D to dogs, while their teeth are in the developmental period, caused the teeth, when erupted, to be defective in structure and improperly placed in poorly calcified jaws. She has also ascertained that certain cereals, notably oats, cause similar effects if a superabundance of Vitamin D is lacking in such a diet.

She and her associates have reported similar results in experiments on rabbits, rats and man. According to her, the type of diet with a sufficiency of Vitamin D that causes the development of sound teeth properly placed tends to prevent dental caries in teeth that have been erupted. This effect, she thinks, may be due to increasing the resistance of the teeth to caries. She places little emphasis on the possibility of variations in salivary composition

and insists that the bacteriological factor is relatively unimportant. McCollum is impressed with the importance of a deficiency of Vitamin D and an unbalanced ratio of phosphorus and calcium as the cause of caries, but is inclined to think that this is brought about by variations in saliva rather than having to do with tooth structure.

Howe, intrigued by the structural changes produced by deficiencies of Vitamin C in developing teeth, especially of guinea pigs as shown by him, Höjer, and others, believes that Vitamin C deficiency is the primary cause of dental caries. Guinea pigs and monkeys kept on diets deficient in Vitamin C, and in some instances in other food principles, developed caries and marked changes in the soft and hard tissues of the dental arches. Boyd and Drain observed a great decrease in the incidence of caries in children whose diet was changed so as to increase the ingestion of all vitamins and mineral containing foods—milk, fresh fruits, vegetables and cod liver oil. Hawkins and his associates emphasize the importance of diets designed to yield a high ash and an increase in the amount of calcium consumed. Bunting concludes that a diet free from refined carbohydrate (cane sugar) tends to inhibit caries to a greater degree than germicidal mouth washes.

The dietary group has given a great deal of attention to tooth structure and the possibility of changing it by modifying nutrition. This implies that tooth composition can be changed after eruption, and is founded on experiments such as those reported by Fish, in which both dentin and enamel of young dogs have been permeated by diffusible dyes from the pulp along definite paths to a degree that led Fish to conclude that there is a circulation of lymph in definite channels in dentin and supposedly also in enamel. However, such experiments are only partially successful in enamel, even in very young animals. In older animals the dyes penetrate the enamel only rarely and in places that may well be defects in it. However, there are those, such as Boedecker, who consider that caries is related to this conception, a disease of vital structures, beginning inside rather than outside of the tooth, and due to some defect of the so-called tooth circulation. Gies injected a dye (trypan blue) into very young dogs; this was deposited in the developing enamel. Although the dye disappeared from all other tissues of the body after the injections were discontinued, the dye that was once incorporated in enamel was never dissolved out as long as the animals lived. This experiment may be considered as crucial, since according to it there can be no circulation in enamel and no exchange of substance in enamel. Enamel can be approached only from the outside and caries is fundamentally a process that concerns that outside surface.

Another illustration of this point may be found in mottled enamel, which is a well known defect of teeth caused by an excess of fluorine ingestion in drinking water during the time that enamel

is being formed. Both Williams and McKay have noted that caries is, at least, no more frequent and apparently appreciably less, in the mouths of the persons whose teeth are in this condition. Furthermore, they call attention to the fact that when decay does occur, it is found not in the mottled enamel defects, but in the usual locations. Such a mottled enamel defect on an immune surface is no more likely to succumb to decay than intact enamel in the same location. It would seem that if there is any type of defective enamel structure that would favor the beginning of caries it would be that found in mottled enamel. Such findings may be accepted as indicating that defective enamel structure, except of the occlusal pit and fissure type, has little or nothing to do with the cause of caries.

The only uniformly successful attempt to produce caries in animals by modification of food is reported by Hoppert, *et al.* They produced caries in rat molars by using coarsely ground corn, the particles being of such a size as to favor impaction between the molar cusps. Caries was initiated on the occlusal surfaces of these molars beneath the impacted particles, regardless of whether the diet was adequate or deficient. Rosebury was able to verify these experiments not only with corn, but also with rice, and noted that the caries incidence was higher in animals on deficient diets, than in animals whose diet was fully adequate. These experiments supply little support to the primary importance of diet in caries etiology, except in so far as it is related to food impaction.

As far as diet is concerned, Rosebury concludes that subtotal reductions in the incidence of caries can be obtained by modifying diet and improving nutrition, although the successful modification is in the form of general correction of all deficiencies, rather than in the replacement of a single specific deficiency. By that he means that in a given group, say of children with a high caries incidence and all on an inadequate diet, generous correction of all dietary deficiencies will result in a marked decrease in the general caries incidence, but in individual members, caries susceptibility will persist and in others the experiment will be only partially successful.

A very convincing example of the effect of a generously adequate diet in all respects is described elsewhere in this work by Dr. Clara Davis, who reports self-selection feeding experiments, extending over a period of nearly six years, with a group of children. The relationship of these studies to the incidence of dental caries is worthy of comment, especially because dental caries had no place in the work as originally planned. The fact that these children had no caries during the experimental period carries great weight. The members of the group whose histories are known to date (four years later) are still without tooth decay.

Those of the nutritional school have, for the most part based their investigations on the hypothesis that variations in suscepti-

bility and immunity could be explained by differences in tooth structure, also that the structure of the teeth, after being completely formed, could be so modified through nutritional channels, as to make them immune to dental caries. Some of these investigators have presented evidence of changes in bones from which they appear, by analogy, to have presumed that similar changes occur in dentin and also in enamel. They have apparently overlooked the fact that evidence must be presented of metabolic change in, or permeability of the enamel through its external surface, to support such a position.

No one should question the fact that nutrition must play an important rôle in susceptibility and immunity to dental caries. There is ample evidence of the effect of diet on the structure of the teeth during their formative period, but it seems probable that its influence for susceptibility or immunity is through the saliva. Investigators who have favored one organism or another as the producer of the acid which causes caries, may all be in a measure correct. The fact that yeast and the acidophilus together, form more acid than either alone, suggests the possibility that several organisms are concerned in the caries process. It seems possible that studies of the relation of variations in diet to the occurrence of caries may be harmonized with the bacterio-chemical findings — that the diet may so effect the saliva as to be the controlling factor, while the microorganisms may be the active cause.

Fosdick and Hansen, of the group at Northwestern University Dental School who are studying this problem, showed that certain chemical tests of the saliva, as indicating the degree of susceptibility to caries, checked very closely with the most critical clinical examination of patients by Blackwell and Bodmer. To the time of this writing more than five hundred patients have been examined and the chemical reports have corresponded with the estimate made as a result of clinical examination in more than 85 per cent of the cases. The significance of this study lies in the fact that the finding of an index of susceptibility in the saliva is of itself evidence that the control of susceptibility is in the environment of the teeth.

One gram of powdered enamel is added to 15 cc. of saliva in a special test tube. The tube is attached to a wheel, which is rotated in a body temperature water bath for four hours. The bacteria in the saliva appear to act on the sugar and carbohydrates to produce organic acids, which in turn dissolve calcium out of the enamel as soon as their concentration is sufficiently high. The amount of calcium going into solution becomes an index of the susceptibility of the individual because it indicates the amount of acid produced and the speed of the change.

This test does not explain how, in dental caries, the acid in the mouth is produced locally and confined to definite areas on the surface of the enamel. The findings are apparently dependent on salivary factors in susceptible cases, which in the mouth so affect

the tooth environment as to favor decay, and in the test tube bring about decalcification of the powdered enamel incubated with the saliva.

Blayney, *et al.*, have reported examinations of stained smears taken from tooth surfaces after these surfaces had been cleaned with a stream of water and dried. They have also examined bacterial growths, attached to the surface of decaying enamel. Scrapings made from all surfaces of the teeth contained microorganisms, but in those made from areas where decay was in progress, diplococci and short rod forms predominate, while these forms are relatively rare in scrapings from other surfaces of the teeth of the same mouth, or from any of the surfaces of teeth of persons immune to dental caries. Therefore, the presence of many of the particular diplococci and short rod forms are considered an index of susceptibility. Here again is an indication that the cause of decay is likely to be found in the environment of the teeth.

Figure 151B is the bacterial picture of a smear taken from the surface of the enamel where decay was progressing, while Figure 151C shows types of growth from an immune surface. The latter



FIG. 151B.

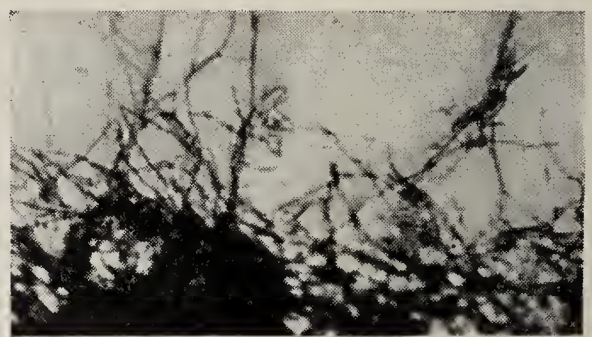


FIG. 151C.

FIG. 151B. Bacteriological picture of scrapings from the surface of the enamel in which the diplococci and the rod forms classify the patient as susceptible to caries. (Blayney)

FIG. 151C. Bacteriological picture of scrapings from the surface of the enamel, which suggest immunity to caries, for the reason that the organisms associated with caries are not present. (Blayney)

contains no organisms indicating immunity; it is of interest only for the fact that the diplococci and rod forms are absent.

This history, briefly as it is written here, shows the steps of the unfolding of our knowledge of this disease process, extending through many years of labor by many individuals, each building upon the discoveries which were made by his predecessors. This work has finally given a complete and exact knowledge of the clinical picture of the beginnings and surface extensions in caries of enamel; the steps in caries of dentin and the nature of it; and the penetration and gradual dissolution of both enamel and dentin. It will also be seen that the work of Dr. Miller was really the finding of the method of the formation of the acids which the older anonymous German writer (1530), Robertson (1835), Regnard (1838), and others, described as being formed by decomposition.

The solution of this problem lies in an adequate explanation of how and by what mechanism the acid is applied and held to the surface of enamel that succumbs to caries. This summary mentions the work of but few of those who have advanced our knowledge of the subject. Many investigators have had a part in it.

GENERAL STATEMENT.

CARIES in its simplest expression consists in a chemical dissolution of the calcium salts of the tooth by lactic acid, followed by the decomposition of the organic matrix, or gelatinous body, which, in the dentin, is left after the solution of the calcium salts. This solution always begins upon the surface, never in the interior. Decay of the teeth is therefore caused by an agent acting from without the tooth, never from within the tooth. It is something extraneous to the tooth, acting upon the surface in the beginning and penetrating little by little into its substance.

In caries of the enamel, the whole substance of the tissue is removed by dissolving out the calcium salts, there being so little organic matrix in the enamel that it will not hang together; consequently a cavity is formed by the simple solution of the calcium salts of which it is composed. The calcium salts are not dissolved completely from without inward in the first instance, but the acid which dissolves them seems to spread, or percolate, into the tissue and the solution goes on as a gradual softening process. The innermost part affected is less softened than the more superficial parts. In the outer portion of the softening area all, or about all, of the calcium salts may be dissolved, while in the inner portion but very little is yet dissolved. In this way the action of the acid progresses slowly from without inward. It first dissolves the cementing substance from between the enamel rods and then attacks the rods. The penetration is in the direction of the rods.

Caries of the dentin is different from caries of the enamel, in that the organic matrix is sufficient in amount and consistence to retain its histological and physical forms after the solution and removal of the calcium salts. With the removal of these by an acid, the dentin will not suffer any change of form. Therefore, the simple solution of the calcium salts leaves a softened matrix in the dentin and does not form a cavity. After the solution of the calcium salts, there is a decomposition of this organic matrix progressing from without inward, breaking it up and finally forming a cavity. Between the solution of the calcium salts and the decomposition of the organic matrix, some little time passes. The two seem never to occur at the same time in any given portion of the tooth, but the calcium salts are dissolved out first and the decomposition of the organic matrix follows later. Therefore, while, after the full development of the carious process, both may be progressing at the same time, the solution of the calcium salts is

always considerably in advance of the decomposition of the organic matrix, leaving a zone of softened material between. There may be a very large amount of material that is soft and spongy, which may be easily cut with a sharp excavator; or, after removing any overlapping enamel that may interfere, a spoon excavator may be passed along the margin of the decayed area, and the whole of it may be turned out in a body, soft enough to be cut with a razor or sharp knife, without injury to its keen edge. Occasionally almost the entire dentin of the crown of the tooth will be softened in this way, while the organic matrix remains intact. This, however, is the exception to the rule. The general rule is that the decomposition of the organic matrix follows fairly closely the removal of the calcium salts.

In the progress of the solution of the calcium salts of the dentin, the tendency is to spread in every direction from the point of penetration through the enamel, and especially along the dento-enamel junction. Then, particularly when the original opening through the enamel has been small, the enlargement of this opening is brought about mostly by what is termed "backward decay of the enamel." This is decay of the inner surface of the enamel that is in contact with the decaying dentin immediately under it, due to the spreading just mentioned. In this case the enamel decays from the inside outward until it is so weakened that it breaks away, enlarging the opening. The rapidity of this backward decay of the enamel is exceedingly variable. Cavities may be wide open early in their progress, or they may remain for a considerable time with a small opening.

The points of beginning and the process by which the structures of the teeth are destroyed are well understood; the fact is definitely established that dental caries always begins by attacking first the outer surface of the enamel.

The dentin is never involved in the carious process until after the enamel has been penetrated. Involvements of the dental pulp by caries are always subsequent to caries of dentin. The functions, possible changes and reactions of these tissues have no bearing on the beginnings of decay.

Therefore, in further studies of the etiology of caries, investigators must be concerned primarily with conditions which have to do with the application of acid to the surface of the enamel in such manner that the calcium salts are dissolved.

Caries affects the teeth of the temporary and permanent dentitions in the same manner. There appears to be no difference in the conditions under which it penetrates first the enamel, then the dentin, and, if neglected, causes inflammation and death of the dental pulp.

CARIES OF ENAMEL

ILLUSTRATIONS: FIGURES 152-183.

CARIES OF ENAMEL is caused by an acid formed by microorganisms under the following conditions: (1.) The organisms are attached to or lie upon the outside of the enamel. They grow and form the acid which causes caries of enamel in that position. They never enter the tissue until the enamel rods are loosened and fall out. The enamel is a solid into which microorganisms can not penetrate. (2.) The enamel rods are cemented together by a cementing substance which dissolves more readily in an acid than the rods themselves, and the first effect upon the enamel is to dissolve out this cementing substance. The general rule is that in decays occurring on the smooth surfaces of the teeth, this cementing substance is dissolved through the entire thickness of the enamel before any enamel rods fall away. In some of the decays occurring in pits, the enamel rods themselves will be dissolved, enlarging the pit. (3.) The decaying spot upon the enamel is always whitened, as the first observable change. This change in color is sometimes not considerable and is very easily overlooked while the teeth are wet, but, when the teeth are dried and examined carefully, the color will be found to be a grayish white, or even very white in some cases, and the outlines are often very clearly marked. (4.) In such spots an explorer is likely to catch if passed lightly over the surface, instead of gliding smoothly as it will on sound enamel. (5.) For these decays to occur, it seems to be necessary that microorganisms become attached to the surface of the tooth, grow there in the form of a colony, attached together in a gelatinoid matrix, or equivalent covering, and produce fermentative decomposition with acid formation at the spot, applying the acid directly to the solution of the tooth. (6.) For this reason, the beginning of caries of the teeth occurs at such points as will favor such lodgment or attachment in which the microorganisms will not be subject to such frequent dislodgment as would prevent a fairly continuous growth. This explains the localization of the beginnings of caries on particular parts of the surface of the tooth.

THE POINTS OF LOCALIZATION ARE: (1.) Pits or fissures in the occlusal surfaces of the bicuspid and molars, in the buccal surfaces of the molars, and sometimes in the lingual surfaces also, and occasionally in the lingual surfaces of the upper incisors. (2.) In the proximal surfaces of all of the teeth. (3.) In the gingival third of the buccal or labial surfaces of all of the teeth, and



FIG. 152A.



FIG. 152B.

FIG. 152A. Plaque growing on the surface of the enamel. The bacteria have grown into the defect in the enamel.

FIG. 152B. Higher magnification of the lamella, or defect, in the enamel, showing the organisms which have grown into it.

rarely in the lingual surfaces also. Ninety-eight per cent of all of the decays that occur in the human teeth are located at the points mentioned.

It may be laid down as a principle that *for caries to begin in the enamel of the teeth anywhere, microorganisms, which form an acid, must be attached to the surface of the enamel in some such way as to prevent the acid which they form from being readily washed away and dissipated in the general fluids of the mouth.*

Under all the observations of the occurrence of decay of the enamel, one cannot conceive of its beginning without the existence of some such conditions. But it is not insisted here that this must

always be by the formation of gelatinoid plaques. A comparable situation may be produced in an artificial way by cementing a band on a tooth, as is done in orthodontia operations, and omitting the cement in a part of the area covered by the band. If such a band remains long on the tooth, caries of the enamel will occur, even when the patient is otherwise immune to caries. This has been a matter of careful experiment by the author.

Figure 152A illustrates a plaque growing on the surface of the enamel, which has been invaded by bacteria. These have also grown into the lamella, or defect, in the surface of the enamel. Something of the structure of the enamel may be seen under the plaque, although most of it has been removed in decalcifying the specimen.

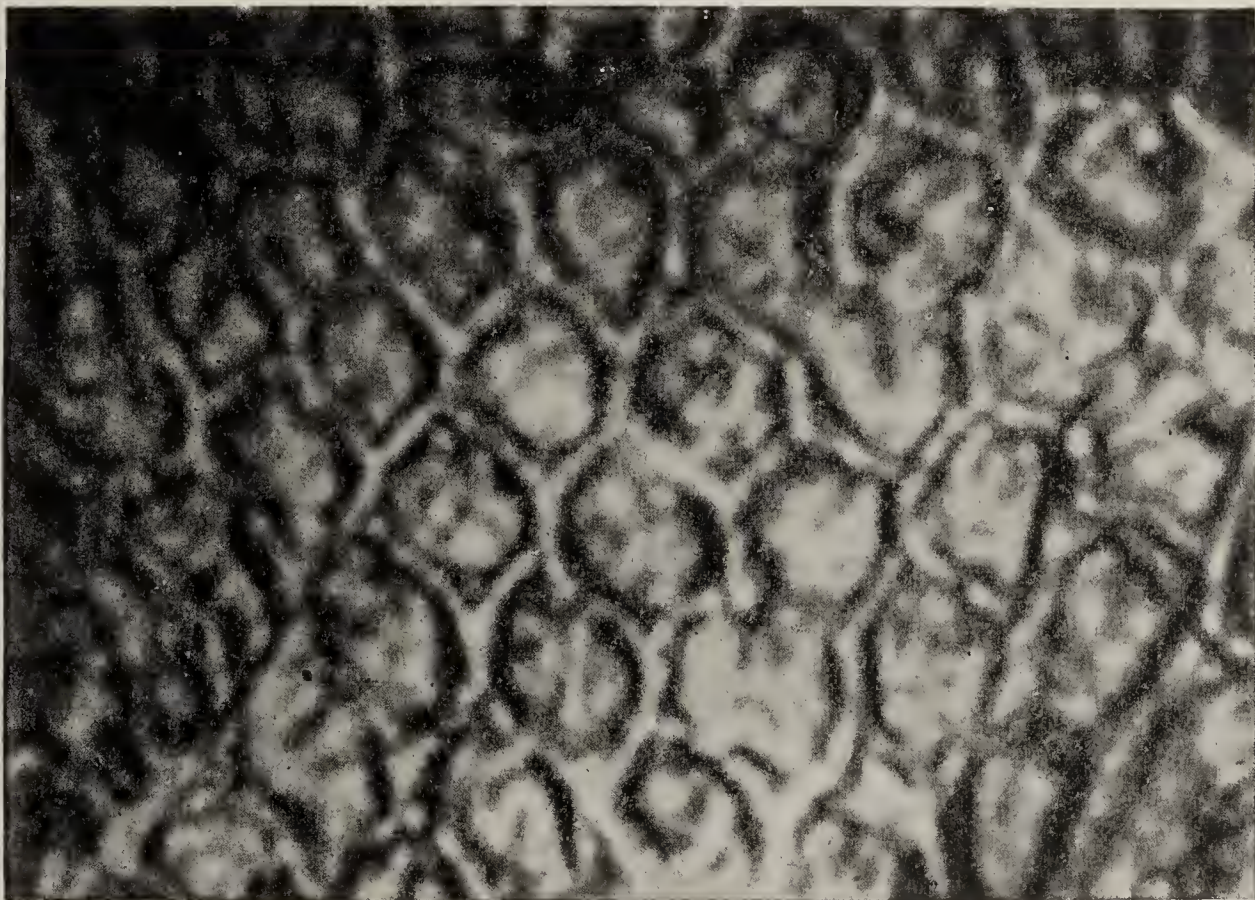


FIG. 153. Ground section of the enamel. Unstained. Magn. $\pm 4000\times$. The angular outline of the cross sectioned hexagonal prisms may be clearly recognized. (Meyer's Histology and Histogenesis. Churchill.)

Figure 152B is a higher magnification of the organisms in the lamella. Normally the surface of the enamel is continuously smooth, except for failures of union at the points or along lines of junction of the lobes of the enamel in its formation. This lamella is a defect in an otherwise smooth surface.

Figure 153 is a ground section of the enamel, cut crosswise of the length of the prisms, to show their hexagonal form. In caries, the cementing substance which holds the prisms together, is dissolved first.

PENETRATION OF ENAMEL IN PITS AND FISSURES.

ILLUSTRATIONS: FIGURES 154-158.

The beginning of caries in pits in the occlusal surfaces of five molar teeth is shown in Figures 154-158. Figure 154 is a "split" bicuspid, which shows a deep fissure, with no decay. This might also be an illustration of a deep pit, as both would look the same in such a section. It is not difficult to conceive of a colony of the microorganisms present in the mouth, establishing itself within one of these pits and growing there, producing acid sufficient to begin the solution of the cementing substance between the enamel rods. These would be covered with debris from foods that are forced in upon them, very completely shielding the acid formed from being washed away by the saliva. It seems to be true that there is a greater intensity of the action of the acid in caries of enamel in this position than in any other, for elsewhere it is very rare to see the carious process extending across the length of enamel rods, or a complete solution of any of the rods before they



FIG. 154.



FIG. 155.

FIG. 154. A split bicuspid with a deep, undecayed pit.

FIG. 155. A very small beginning of caries in a pit.

are loosened from the dentin. Caries usually follows accurately the length of the enamel rods in the penetration of the enamel in all axial surface positions. But in beginning decays in pits, occasionally the dissolution progresses across the length of the enamel rods. This is shown in Figure 157 particularly. The pit has been enlarged at its deeper part by the solution of the enamel rods about its walls. This occurs frequently and indicates that the locality is more completely isolated, or less disturbed by solution of the acid in the oral secretions than elsewhere, and therefore reaches the highest percentage of acidulation. In this position the rods themselves are often dissolved even before the carious process has

extended into the dentin. The spreading across the enamel rods is, however, often more apparent than real, for the enamel rods about pits are all inclined toward the pits. Figure 158 is a photograph of a split temporary molar tooth. The cut was made directly through the distal pit, which shows a typical area of decay of the enamel, with slight involvement of the dentin. The cut was made a little to one side of another pit near the mesial margin of the occlusal surface. There is also a mesio-occlusal cavity which had been restored with copper cement.

It will be noted also, in the examination of these illustrations, that there is no sign of the carious process on the surface of the enamel outside the pit. It is confined exclusively to the walls of the pits and usually to the deeper part. Occasionally pits that are gradually narrowed from surfaces much inclined, or in a funnel



FIG. 156. A more extensive involvement of the enamel, in which the triangular form of the section through the conical area is very definite.

shape, will show caries extending a little out of the pit proper, but this is unusual. This absence of superficial extension is because the surfaces immediately about the pits are kept clean, or reasonably clean, in the process of mastication; and further, because, if micro-organisms were growing in such a position, they would be subjected to continual washings by the saliva, which would dissipate the acid formed. *Therefore, decays of the enamel, beginning in pits, are in the form of a cone, having the base on or toward the dento-enamel junction, and the apex toward the surface.*

If there are fissures in the enamel along the lines of the grooves, caries will occur in the same manner as in the pits. As a rule, pits are deeper than the fissures, which extend from the pits in various directions. A groove is smoothly rounded and so shallow that its surface is cleaned in the process of mastication. When the lobes of enamel have failed to join perfectly, so that food will be packed into the crevice, or fissure, conditions as to the beginning

of caries are the same as for a pit. *Therefore, in the preparation of cavities for the treatment of pit and fissure decays, it is necessary that cavities be extended along the lines of the fissures so that the margins may be placed in positions that are cleansed in mastication. This is called extension for prevention of recurrence of decay.**

PENETRATION OF ENAMEL IN SMOOTH SURFACES.

Conditions are different on the smooth surfaces of the teeth, as, for example, the proximal surfaces. Here there is no pit or fissure, depression or fault in the enamel. It is a smooth, rounded surface. The teeth round together normally in such a way that the interproximal gum tissue fills the space to the contact point,



FIG. 157.



FIG. 158.

FIG. 157. In this case some of the enamel rods have fallen away and there is slight involvement of the dentin.

FIG. 158. A split temporary molar, which shows a typical penetration of a distal pit — to the left in the illustration. The cut through the tooth is a little to one side of the center of the other pit.

and the contact point proper is fairly well rounded in most cases. While this condition continues perfect, there is no place for the growth of a colony of microorganisms upon the proximal surfaces, but, whenever the gum tissue fails to fill this space completely and a little opening is left to the gingival of the contact point, it forms a harbor or nidus where microorganisms may lodge and begin their growth. It is probable that by this growth alone they may have something to do with forcing the gum tissue a little bit farther away, and, if they are sufficiently protected from washings

*This application of the term "extension for prevention" to the preparation of pit and fissure cavities is new. It seems justified as indicating the reason for the cutting of these cavities to positions where smooth margins may be made.

by the saliva, the acid formed by them may act upon the calcium salts of which the enamel is formed.

The location of such a colony, and therefore the point at which proximal decays begin, is governed by the forms of the proximal surfaces of the teeth in their relation to the contact point. The incisors and cuspids are narrow labio-lingually at the position of their contacts; proximal decays therefore usually begin at a single point or small area to the gingival of the contact. The bicuspid are somewhat broader bucco-lingually, so that a slight recession of the gingiva may permit of one or more points of beginning; usually only one on the mesial surface of the first bicuspid, and frequently two or more on the distal of this tooth, also on both proximal surfaces of the second bicuspid. The molars, being broader than the bicuspid, will more often show several points of beginning in a row parallel with the margin of the gingiva.

Smooth surface decays on labial and buccal surfaces also begin at one or more points in the enamel close to the margin of the gingiva, in areas which are not kept clean. These spread on the surface mainly toward the mesial and distal angles of the teeth, keeping close to the margin of the gingiva.

In all smooth surface decays the penetration of the enamel is in the direction of the rods, the cementing substance being dissolved, usually through the full thickness of the enamel, before any of the rods fall away. The deepest and the first complete penetration is always in the position of the point of beginning; the penetration is less deep in the surrounding area, corresponding to the spread of the colony of microorganisms on the surface of the enamel. *Therefore, decays of enamel, beginning on smooth surfaces, are generally in the form of a cone, having its base at the surface of the enamel, and the apex toward the dentin.*

Where there are two or more points of beginning, it is obvious that there will be a corresponding number of cones, and the depth of penetration of the apices of these cones will correspond with the length of time that colonies of acid-forming organisms have grown upon the several surface areas. As time passes the cones may coalesce and the apices will become broader as full penetration of surrounding areas occurs. Eventually, the area of complete penetration of the enamel may correspond to the full area of surface attacked by the acid.

In the consideration of the beginning of caries of the enamel with reference to treatment, it may be stated, as a fundamental proposition, *that the nidus of each beginning of caries will be found at that particular point on the surface of the tooth attacked, or that may be attacked in the future, that offers the best position for the lodgment and undisturbed growth of colonies of microorganisms.*

These colonies and the caries will spread superficially on the

surface to those lines where their farther spread is limited, (1) by the position of normal gum tissue; (2) by friction of mastication by excursions of food through the embrasures; or (3) by artificial cleaning. When this nidus is destroyed by the falling away of the enamel rods because of caries of the dentin, before such spreading has reached its limits, as often happens, the conditions are so changed that the growth of colonies on the surface of the enamel is prevented. This at once limits further superficial spreading buccally and lingually on the lines thus far illustrated. Further superficial spreading gingivally, may or may not occur, as will be illustrated later. The lateral superficial spreading seems to be stopped by the frequent catching of stringy foods on the roughened area and the interference with the spread of the surface growth in their removal, or by the frequent forcing of these lodgments further to the gingival at each meal time. In many of the cases the loss of the central and best attachment of the organisms may of itself be sufficient to produce this effect. However this may be, much careful observation shows that after the opening of the cavity this stoppage occurs in many cases. But if a good and perfect restoration of the original form is made, without reference to the possible further spreading of the growth of colonies of microorganisms, such restoration will reestablish the nidus for growth and this will recur on the restoration and extend to the full limit that it might have done if the original nidus had not been destroyed, thus reestablishing caries beyond the margins of the restoration. In this way, a mechanically well-made restoration of the original contour, without reference to the superficial spreading of caries, may become the cause of wider spreading of decay on the surface than would have occurred without it. Further, in practice, it will be found that when proximal restorations are made without separating the teeth to gain room for finishing to the full original contour, but necessarily losing a little of the original mesio-distal breadth, the area of near approach of the proximal surfaces is increased, the embrasures are made shallower and the opportunity for recurrence of caries at the bucco-gingival and linguo-gingival angles of restorations is increased.

SUPERFICIAL SPREADING OF CARIES IN PROXIMAL SURFACES OF BICUSPIDS AND MOLARS.

ILLUSTRATIONS: FIGURES 159-169.

The superficial conditions as seen in the proximal surfaces of the bicuspid and molars are, in some respects, different from those of the incisors. Examination of the whitened outlines seen upon the surfaces of the teeth before any enamel rods have fallen away, shows that these decays take certain definite forms by reason of spreading, and often start at several points instead of one central

nidus. A knowledge of these forms and the reasons for them, is of great importance in the treatment of caries.

The group of illustrations, Figures 159-162, inclusive, shows the principal varieties of form produced by the spreading bucco-lingually of beginning decays in the proximal surfaces of the bicuspid. This tendency is practically the same in the molars, as seen in Figure 164. These may be confined to a round spot, as is often the case in the incisors, as shown in Figure 177, but the more general tendency is to spread buccally and lingually from the beginning point. This is shown progressively in the different pictures of this group and illustrates the common tendency of caries



FIG. 159.



FIG. 160.



FIG. 161.



FIG. 162.

FIGS. 159, 160, 161, 162. Photographs from four bicuspid teeth with superficial proximal decays. To show distinctly the cemental line, the first two have been stained slightly with eosin, which does not stain the enamel. The four teeth have been arranged to show progressively the disposition of caries to spread bucco-lingually on the proximal surfaces of these and the molar teeth. While decay is apt to begin first just to the gingival of the contact point and is confined between that and the free border of the gum occluso-lingually, it is free to spread bucco-lingually as far as the sweep of food through the embrasures, formed by the rounding of the angles of the teeth away from each other will allow. In Figure 159 the area bucco-lingually is very narrow. In Figure 160 a little broader, and in Figure 161 it reaches fully to the embrasures. In Figure 162 something of the disposition of decay to begin at numbers of small points along this bucco-lingual line, is seen.

of the enamel to spread in these particular directions. Occasionally the tendency to spread gingivally is seen, as is illustrated in Figure 163. The cause of this will be more explicitly discussed later. Spreading occlusally does not ordinarily occur, because that part of the surface from the contact occlusally is cleaned by mastication. However, decays that begin much to the gingival of the contact point may spread occlusally. It will be readily seen that if these teeth are cut horizontally through the decay, the area of decay presented in the section will be broad, while in vertical sections it will be narrow.

In the group of illustrations, Figures 165-169, inclusive, horizontal sections are shown illustrating the conditions from that view. The teeth were cut horizontally through areas of decay which, in surface appearance, were similar to those in Figures 159-164. These have the additional value of showing both the spreading buccally and lingually and at the same time the penetration of the enamel along this line in its relations to the individual teeth as a whole. In three of the four figures, the decay

has spread across the proximal surfaces to the rounding of the angle which opens the embrasures, both to the buccal and to the lingual, practically before decay of the dentin has begun.

Figure 165 is part of a horizontal section of a molar showing a beginning decay with very slight penetration of the enamel. Figure 166 is from a mouth with thick necked, squarely built teeth, in which the lingual surfaces of the second bicuspid are equally broad with the buccal, and with very flat proximal surfaces. On the distal, the decay reaches fully from angle to angle. It is easily seen in this that there were two nearly equal beginning points which have run together on the surface, but are still divided in the deeper part. On the mesial surface, it is noted that the original beginning was much to the lingual at the point where

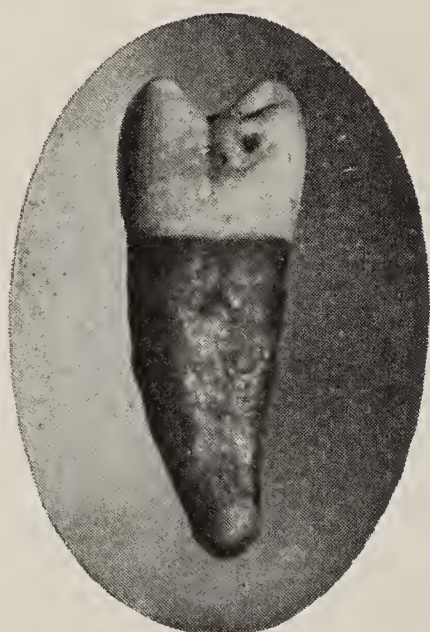


FIG. 163.



FIG. 164.

FIG. 163. A bicuspid in which there is seen a disposition to broader spreading occluso-lingually. Sometimes this is very extensive, as will be illustrated later.

FIG. 164. An upper molar in which is seen the same disposition to spread bucco-lingually as occurs in the bicuspid. This form of spreading is common to the bicuspid and molars. In this case the enamel rods have fallen out of a small area at the point of first beginning.

whitening completely penetrates the enamel for a little space. The spreading is toward the buccal and toward the lingual from that point. It spreads a little around the mesio-lingual angle but does not reach the mesio-buccal angle. It is easy to determine by this that the position of the first bicuspid was abnormally to the lingual of the line of the arch. This gave the opportunity for decay in this unusual position. It was prevented from reaching the mesio-buccal angle by the cleaning of this part by excursions of food crushed along this portion of the surface in mastication.

In Figure 167 there has been an unusual number of very small beginning points forming a line which had just fused together by the superficial spreading. It is particularly interesting from that fact. The tooth seems out of form for the reason that the decayed

side was cut farther from the occlusal surface than the sound side. Figure 168 shows an area of decay on the distal surface of a molar that is of the more regular type, showing a solid advance of decay of the enamel, except as it thins out toward the angles of the tooth.

Many of the beginning decays observed in the proximal surfaces are of much less extent than in these illustrations. Indeed, the general rule is that the enamel rods have fallen away in the central area quite a little before the spreading has reached its bucco-lingual limit. In such cases, the spread of decay in the dentin,



FIG. 165. Horizontal section of a molar with a beginning proximal decay that has penetrated the enamel very slightly. The area is broad bucco-lingually, but very narrow occluso-gingivally.



FIG. 166.

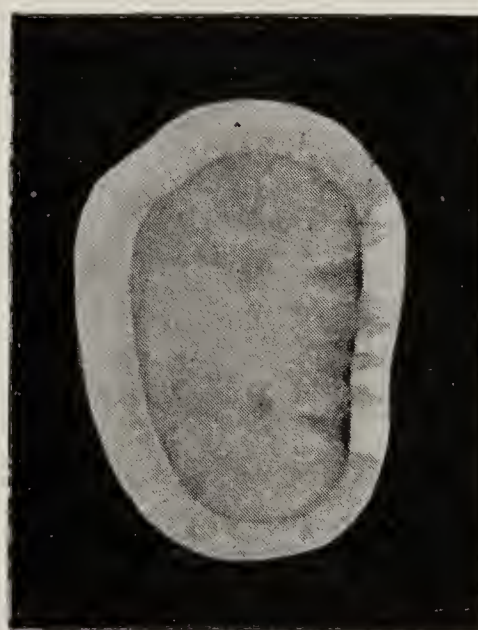


FIG. 167.

FIG. 166. A cross cut of a bicuspid crown showing decay on both the mesial and distal surfaces. On the right side of the picture the spreading of decay is from angle to angle; indeed, somewhat around the curve on the lingual. Also the decay is seen to have begun at several points along the line from the buccal to lingual. Some solution of the calcium salts of the dentin is in progress, though no enamel rods have fallen away. A broad area of decay is shown in such an illustration, because the cut is along the length of greatest spreading on the surface. A section lengthwise of the tooth would show a narrow area of decay. On the left margin of the illustration, the area of decay is not central because the first bicuspid was in lingual occlusion. Hence the anomalous position of the beginning of decay.

FIG. 167. A cross cut of the crown of a bicuspid with a decay that has begun at a number of points, which have penetrated the enamel separately, leaving some areas of sound enamel between them at the time of extraction, but on the surface all had united.

along the dento-enamel junction, backward decay of the enamel and breaking away of enamel, so change the conditions as to stop the superficial beginnings in the enamel. Figure 169 sufficiently illustrates this. In this case, the spreading bucco-lingually is not yet great, but the decay has penetrated the enamel at two minute points, and solution of the calcium salts of the dentin has begun. In a little more time the enamel rods would have fallen away from all of the central part of the area and so changed the conditions that the superficial spreading of caries would have been prevented by the elimination of the nidus of attachment for new colonies of organism.



FIG. 168.

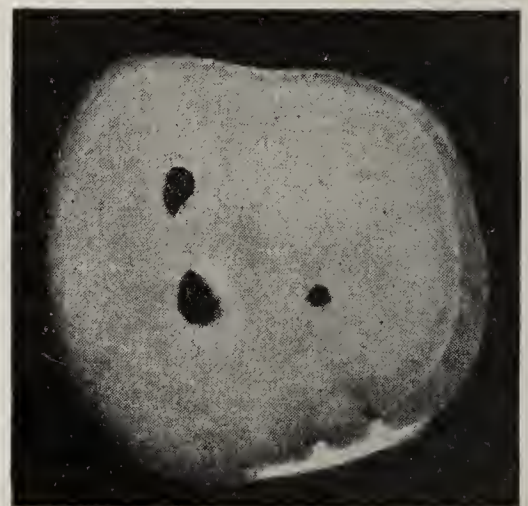


FIG. 169.

FIG. 168. An upper first molar cut across the crown, showing a solid area of caries of the enamel stretching bucco-lingually from angle to angle of the distal surface, which has just reached the dento-enamel junction at one point. One should note especially the thinning out to the surface of the decay of enamel rounding slightly toward both the buccal and lingual angles, and the amount of sound enamel that would have to be removed in order to remove the last of the carious enamel in the preparation of such a case for a restoration.

FIG. 169. An upper first molar with a less extensive decay of the enamel, which has reached the dentin at two points. In this case the beginning of the decay was much further toward the gingival line than usual, and the enamel is very thin.

A CLOSER EXAMINATION OF THE INJURY TO THE ENAMEL.

ILLUSTRATIONS: FIGURES 170-171.

Before studying further the cause of the localization and tendency to the spreading of caries in particular directions on the surface of enamel, it may be well to examine more closely the injury produced. For the illustration of this, the photomicrographs have been taken from thin sections of decays beginning in the enamel that were in every way similar to those shown in other illustrations, but with an amplification that is sufficient to display the condition of the tissue. Special preparation of the tissue has been made to show the removal of the cementing substance from between the enamel rods by filling the spaces left with common



FIG. 170. A photomicrograph showing a portion of a section of enamel in which caries has progressed part way through its thickness. E. Enamel that is perfect. X. Decayed enamel. The decayed portion of the enamel has been filled with yellow shellac which has taken the place of the cementing substance dissolved out from between the rods and takes dark. The sound enamel being solid does not absorb the shellac. The plan of doing this is given in the text. In this particular section not much of the structure of the enamel can be seen in the undecayed portion.

yellow shellac, which takes dark in the photomicrograph. In Figures 170, 171, which are both from cross sections, but from different teeth, the darker portion marked with the letter x is the injured enamel, while that portion marked by the letter E is uninjured. In each case the border line between the injured and uninjured tissue is dark. The adjacent undecayed enamel does not absorb the shellac and remains white. In Figure 170 the sound enamel shows scarcely a trace of structure, while in Figure 171 the enamel rods in the sound tissue are very well brought out. In both specimens the area decayed is also sharply divided from

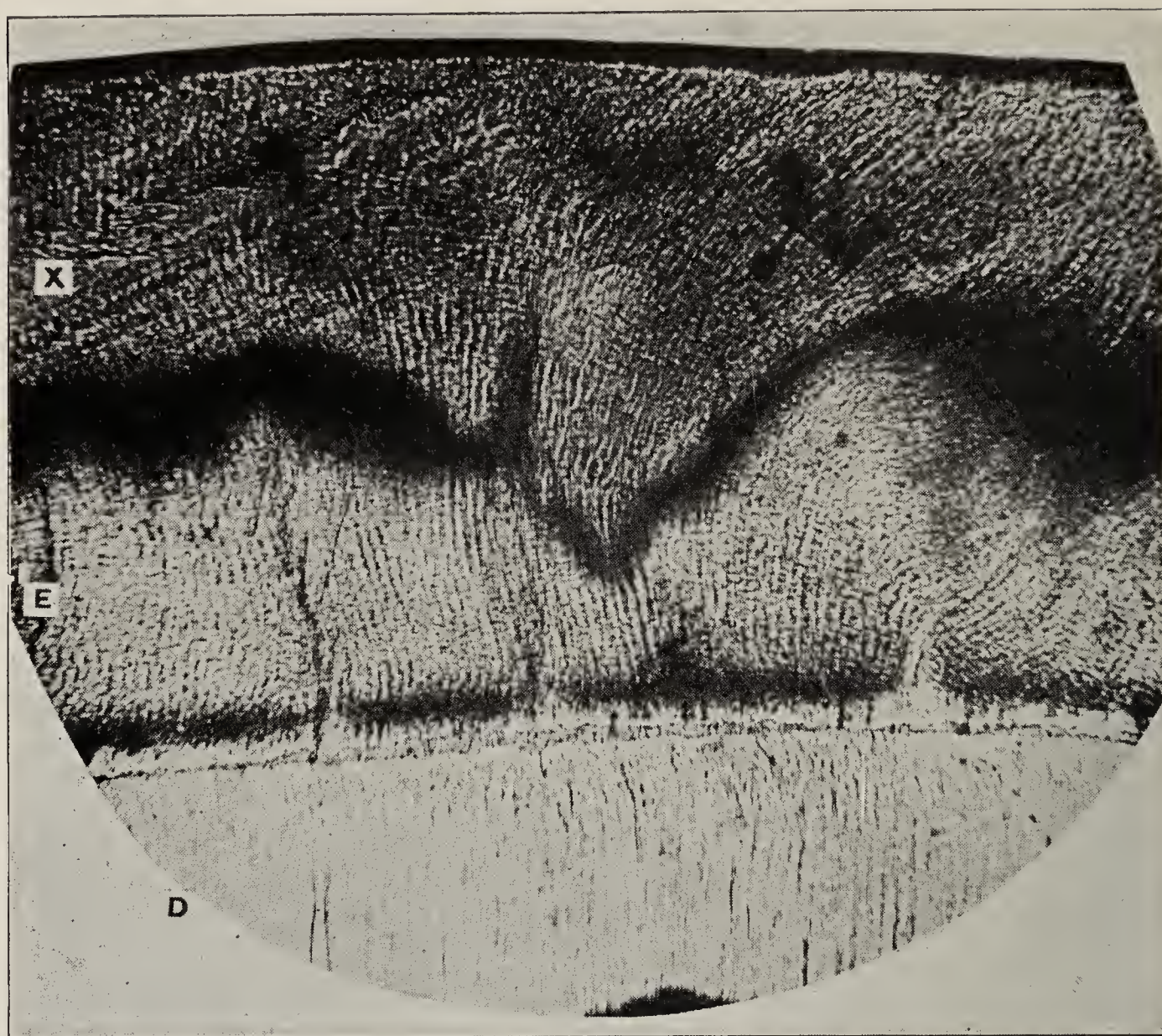


FIG. 171. A photomicrograph of enamel in which caries has made considerable progress. D. Dentin. E. Enamel. X. Cariouss enamel. The dento-enamel junction is seen between D and E. In this case the enamel rods appear fairly well in the sound enamel and a considerable irregularity in their course may be observed. The decayed area is filled with yellow shellac. The enamel rods appear smaller in the decayed area.

the normal enamel by a broad, dark band, which occurs in many specimens in which no shellac has been used, as will be seen in most of the photomicrographs of decayed enamel in this volume. If these illustrations are carefully examined as to the comparative sizes of the enamel rods in the undecayed and the decayed areas, it is found that they are much reduced and more slender in the decayed area. When the solution of the enamel rods has gone a little further than shown in these illustrations, the whole structure falls to pieces at a touch, and most generally through the whole thickness of the enamel at once. This seems to be due to the complete loosening of the ends of the rods from the dentin. In unprotected places, however, such as decays of buccal or labial surfaces, and decays of proximal surfaces in which there has been interference by the crowding of food through the contact, a number of cases are found in which the outer ends of the enamel rods are broken before the enamel has been penetrated. In the greater number of cases, however, in any of these positions, a considerable solution of the dentin has occurred by the acid which has pene-

trated through the enamel, before any enamel rods have fallen away. This is well attested in many of the illustrations presented.

THE RELATION OF THE OCCLUSION TO THE LOCALIZATION OF CARIES.

ILLUSTRATIONS: FIGURES 172-176.

A study of the forms of the teeth in their relations to each other as they stand in the arch, and their uses in the mastication of food, shows that there are certain points or areas of their surfaces that are comparatively free from rubbing, or abrasion, while much the larger part of the tooth surface is exposed to friction and washings by the fluids in the mouth. The occlusal surfaces particularly are exposed to severe friction in the mastication of food. The lingual surfaces, both above and below, are exposed to the friction of the tongue and of the food that is forced over them during mastication, so that these surfaces are fairly well cleaned. The buccal surfaces are less exposed to friction during mastication and to washings by the saliva during the motions of the tongue, cheeks and lips, particularly in their gingival thirds. The proximal surfaces are shielded from friction and from washings by the saliva by the contact and area of near approach of these surfaces with adjoining teeth. Decays beginning in the depths of the pits of the occlusal surfaces have no opportunity to spread upon the surface of the enamel, for the reason that these surfaces are so continually cleaned by abrasion in mastication and by washings by the saliva. It seems to be for these reasons, principally, that decay does not spread superficially upon these surfaces. It is prevented by the forms and the uses of the teeth. It seems to be purely these local conditions that are the basis of the strict localization of the beginnings of dental caries in certain positions in the enamel to the exclusion of all others. These are physical conditions controlling lodgments of debris. They are also physical conditions promoting the retention of anything which may become attached to the enamel in these positions.

EXTENSION FOR PREVENTION.

If the relations of the proximal surfaces of the different teeth to each other are examined, it will be observed that various forms of contact and near approach of these surfaces serve to shield certain areas from all friction from mastication and in which the deposit of a very little gummy material will shield colonies of microorganisms from all washings by saliva. This may be studied in the illustrations. Figures 172, 173, show the buccal and the occlusal surfaces of the upper bicuspid and molars as they stand in the arch. The teeth are in the same relation to each other in each figure. The soft tissue filling the interproximal spaces has been removed in order that the forms of these may be better seen.

In studying the buccal view, it will be seen that the points of near approach of surfaces are very narrow and rounded in form from occlusal to gingival, so that the actual touch point of unworn teeth is very small, like that of two marbles coming in contact, while, in the view of the occlusal surfaces, the points of near approach of the teeth to each other are shown to be very much broader in the bucco-lingual direction.



FIG. 172.



FIG. 173.

FIGS. 172, 173. Photographs of the upper molars and bicuspid of the right side in their normal relations to each other, with the soft parts removed to show the interproximal spaces, interproximal contacts and areas of near approach of the surfaces of the teeth. The two pictures taken together, the first showing the buccal surfaces and the second the occlusal surfaces, give a good view of the interproximal spaces, breadth of contact occluso-lingually, and breadth of near approach of surfaces bucco-lingually with their variations. They also show the openings of embrasures buccally and lingually, with their variations in depth.

The decays in the proximal surfaces of the bicuspid and molars, in Figures 159-164, inclusive, correspond in form with the areas of near approach of the surfaces of the teeth so exactly and so constantly that these relations must be considered as cause and effect, controlling both the localization and superficial form of the beginning and spreading of caries in the enamel. To gain correct

expressions of this, the study must be confined to those beginnings of decay that have not yet penetrated the enamel, or in which the enamel rods have not yet fallen out, for, with the breaking away of the enamel after decay of dentin has begun, the particular form of the beginning upon the surface of the enamel is often quickly lost.

A study of the forms of the surfaces making the contact of different teeth as seen in the occlusal view, will show great variations in the bucco-lingual length of near approach of these surfaces. The area of near approach of surfaces is much greater bucco-lingually between the second bicuspid and the first molar than that between the two bicuspids. Therefore, a beginning decay on the proximal surface of the second bicuspid or first molar will spread wider bucco-lingually than a similar decay on the mesial surface of the second bicuspid or the distal surface of the first



FIG. 174. Photograph of the teeth in normal occlusion, including the alveolar processes and bones. This is given as a study of the normal relations of the cusps of the teeth of the upper arch to the embrasures of the lower, and the relations of the cusps of the lower teeth to the embrasures of the upper. It will be seen that among the bicuspids and molars this relation is constantly such as to force food through these embrasures in chewing.

bicuspid. The differences in this respect among the teeth of the same mouth, and especially among the teeth of different mouths, are very great.

As this study of forms of contact and near approach accounts for the differences seen in forms of decay in this respect, as shown in illustrations given of the bicuspids and molars, Figures 159-164, inclusive, it also accounts for the differences shown of the breadth of decay in illustrations made from cross sections of the teeth, Figures 166-169, as compared with sections made lengthwise of the teeth. A section made lengthwise of a tooth may show a very

narrow injury to the enamel, while if the section were made cross-wise of the tooth, cutting through the length of the injury, it would show a very broad beginning of decay. This will be seen more clearly by a comparative examination of sections cut in these different directions which show the great difference in the form and extent of the injury to the enamel. This will be appreciated by comparing the cross sections, Figures 166-169, with the photographs of split teeth, Figures 197, 198, 201, 205.

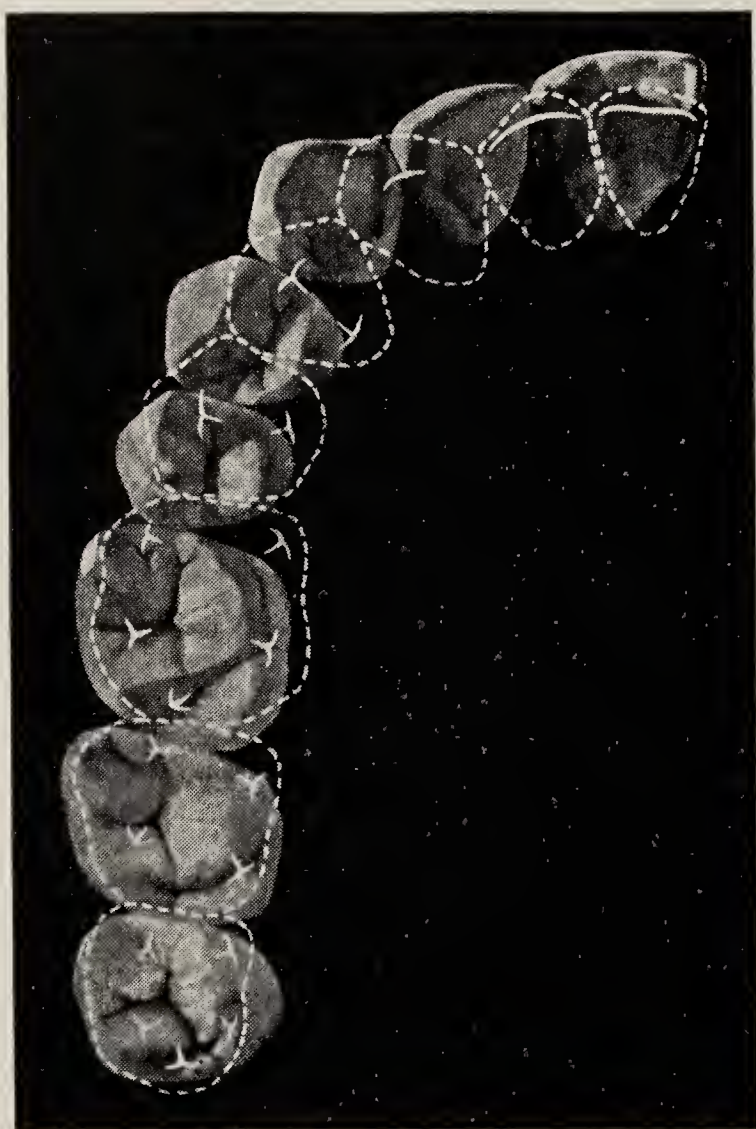


FIG. 175.

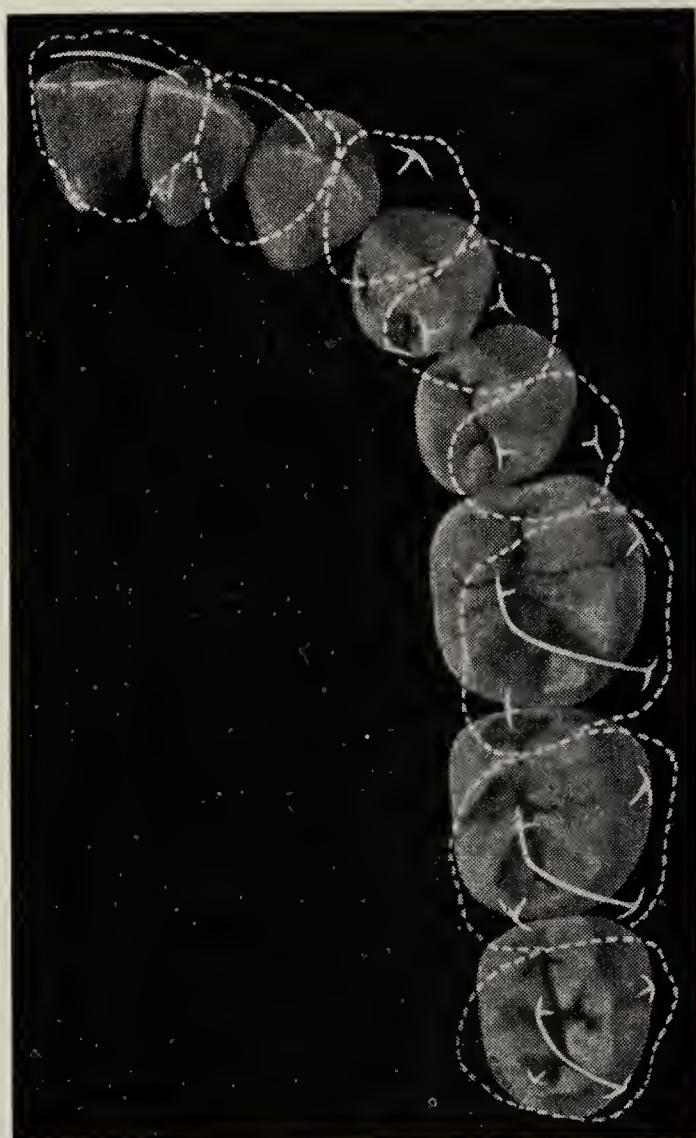


FIG. 176.

FIG. 175. An occlusal view of the upper teeth of one side of the arch with the outlines and cusp positions of the lower teeth superimposed in the positions they occupy when the mouth is closed in the resting position.

FIG. 176 is a similar view of the occlusal surfaces of the lower teeth with the positions of the upper teeth superimposed.

Illustrations by Sheldon Friel; *Int. Jnl. Orth., Oral Surg. and Radiog.*, Vol. 13, 1927.

THE EMBRASURES, or the openings formed on the buccal and on the lingual by the rounding of the surfaces of the teeth away from each other, vary greatly in depth among the different embrasures of the same mouth, and particularly they vary in depth among the teeth of different persons, owing to the forms of the teeth and the form and prominence of the proximal contact points. It must be remembered that in normal conditions in young people, the interproximal space is filled with gum tissue to, or very nearly

to, the contact point, and that the gum tissue arches up to this from the buccal and the lingual. In studying this, it will be found that the beginning of decay is close to the line of the margin of this arch of gum tissue, and it does not spread to the gingival unless the gum tissue has been pushed away by lodgments of food between the teeth. Therefore, unless lodgments of food have occurred, the forms of beginning decay will retain that narrowness from occlusal to gingival that has been shown, and the spreading bucco-lingually will generally coincide with the length of the close approach of the surfaces to each other.

In view of the fact that surface extensions of decay do not involve the enamel under the margin of healthy gum tissue, it is obvious that gingival margins of proximal cavities should be placed in these immune areas. *Therefore, in the preparation of cavities in the proximal surfaces of the teeth, the gingival margin should be placed under the margin of the healthy gum septum. This is extension for prevention of recurrence of decay.* This reference to the healthy gum septum applies to its usual condition, in which a considerable portion of the enamel to the occlusal of the cemental line is covered by the septum. Conditions of extreme recession of the gums will be considered later.

Under normal conditions there is a provision of very considerable importance in the relation of the cusps of the upper teeth to the embrasures between the lower teeth, and, vice versa, in the arrangement of the cusps of the lower teeth with respect to the embrasures between the upper teeth. This may be seen in Figure 174. Beginning with the third molars, the cusp of the lower third molar occludes directly under the embrasure between the second and third molars of the upper jaw and will force food through that embrasure especially, while the second molar of the upper jaw has its distal cusp over the embrasure between the second and third molars of the lower jaw and will force food through it. This arrangement is repeated among all the molars and bicuspids. The result is that, in mastication, the food is caused to run through the embrasures by the crushing action of the teeth. The effect of this arrangement will perhaps be better understood by comparison with the occlusal surfaces of the same teeth as seen in Figures 175, 176, noting carefully the forms of the individual embrasures and their variations. This abrasive action by the food is increased by the cheeks and tongue also pressing upon the food laterally, causing it to be forced down over the buccal and lingual surfaces of the teeth, and more particularly through these embrasures. In fact, this lateral pressure on the food is an endeavor to hold it between the occlusal surfaces of the teeth. The result is that the food is forced through the embrasures especially, causing it to rub the angles of the teeth from their occlusal surfaces to the gum margin.

This abrasive action of the food serves to limit sharply the distance that attached colonies of microorganisms may spread

toward the buccal or lingual angles of the teeth because of their removal from the surfaces by the passing of food through these embrasures during mastication. It also explains another fact of great importance in the treatment of caries of the teeth by restorations, for it was found by actual count of ten thousand persons examined that only in about one case per thousand, decay was found to have spread upon the surfaces of the teeth across the angles. *Therefore, in the preparation of cavities in the proximal surfaces of the teeth, the buccal and lingual margins should be placed beyond the area of liability to surface extensions of decay and within the areas of immunity toward, or near to, the angles of the teeth. This is extension for prevention of the recurrence of decay.*

CARIES NEVER BEGINS ON THE ANGLES OF THE TEETH nor spreads superficially past these angles in any case in which the teeth are in normal relations and the person is making active use of them in mastication. All of the cases of such spreading that have been observed in twenty-five years of close study of this point, have been in persons who have practically ceased to use their teeth in the mastication of food because of some interference. This has generally been sensitiveness of exposed pulps in decayed teeth, and at the same time the persons had become entirely careless as to artificial cleaning. The study of this phase of the spreading of caries and its limitations is of the utmost importance. The student and the practitioner should be continually studying the mouths of their patients, mapping out carefully areas of spreading decay and their normal limitations under the conditions which are found. This will serve as a guide of great importance in the preparation of cavities in such forms as to prevent recurrence of decay about cavity margins.

PENETRATION OF ENAMEL IN PROXIMAL SURFACES OF INCISORS AND CUSPIDS.

ILLUSTRATIONS: FIGURES 177-183.

The differences in the beginnings, surface spreading and penetration of the enamel in proximal surfaces of incisors and cuspids are due to the differences in the forms of these surfaces as compared with the proximal surfaces of the bicuspid and molars. *The rules of extension for prevention in the preparation of cavities are the same; the gingival margin should be placed under the margin of the healthy gum septum, and the labial and lingual margins should be placed beyond the areas of liability to surface extensions of decay and within areas of immunity toward, or near to, the angles of the teeth.*

Figures 177-180 constitute a group showing the manner of the penetration of enamel by caries in the proximal surfaces of incisors, as may be well seen in split teeth with an ordinary hand magnifier.

Figures 181-182 illustrate the same thing in photomicrographs produced by low powers of the microscope. The first beginning of decays upon these surfaces is often small, as is shown by Figure 177, a photograph of the distal surface of a central incisor with the spot of beginning decay very exactly in the position mentioned, just to the gingival of the contact point. This was a very white spot on a tooth that was rather dark in its general color, so that the engraver has been able to show it very distinctly. The question may be raised here, by what power, circumstances or condition has the action of the acid been confined to this one small spot, while all the rest of the surface of the tooth is free from any action of the acid?



FIG. 177.



FIG. 178.

FIG. 177. Caries of enamel. A white carious spot on the distal surface of a central incisor. It has very sharp and definite outlines, though not very regular.

FIG. 178. An incisor removed for a girl nine years of age, split through areas of decay and showing the broad pulp chamber of that age. A decay in the mesial surface had destroyed the pulp. In the distal surface there is an area of decay in the enamel, which superficially, was similar to the one shown in Figure 177, but not so white. This is very typical of the form of these smooth surface decays of enamel in its conical shape, with the broad base of the cone on the surface of the enamel and the apex of the cone toward the dento-enamel junction. In this case the apex of the cone has just penetrated the enamel. A little solution of the lime salts of the dentin has begun by the percolation of acid from the surface through the thickness of the enamel. There is a delicate hyaline zone fringed with shade, which streaks away toward the pulp.

Figure 178 is a photograph from a central incisor that has been split mesio-distally through a white spot upon its distal surface, but little larger than the one shown in Figure 177. In this the penetration into the enamel was in the form of a somewhat flattened cone, or a cone with a broad base on the surface of the enamel, with the point just reaching through to the dento-enamel junction. In caries of the enamel more highly magnified, to be illustrated later, the solution of the cementing substance between the enamel rods follows directly the length of the enamel rods and continues spreading upon the surface of the enamel from a spot of much smaller beginning than that now seen. At the central beginning point, or nidus, the effect of the acid has reached through

the enamel to the dentin and is beginning to dissolve it, while the depth of penetration is less and less about that central point in every direction, until it is reduced to quite a thin edge at the surface of the enamel. This is characteristic of beginning decays in enamel upon all of the smooth surfaces of the teeth. They begin at a central nidus, or beginning point, and spread sometimes in every direction, but generally spread most in some particular directions from that beginning point, as will be described later.

Figure 179 is from a photograph of a lateral incisor, cut mesio-distally, with a cavity in the distal surface. This was a very white decay and shows to advantage. It will be seen that there has been considerable spreading both ways from the central nidus,



FIG. 179.

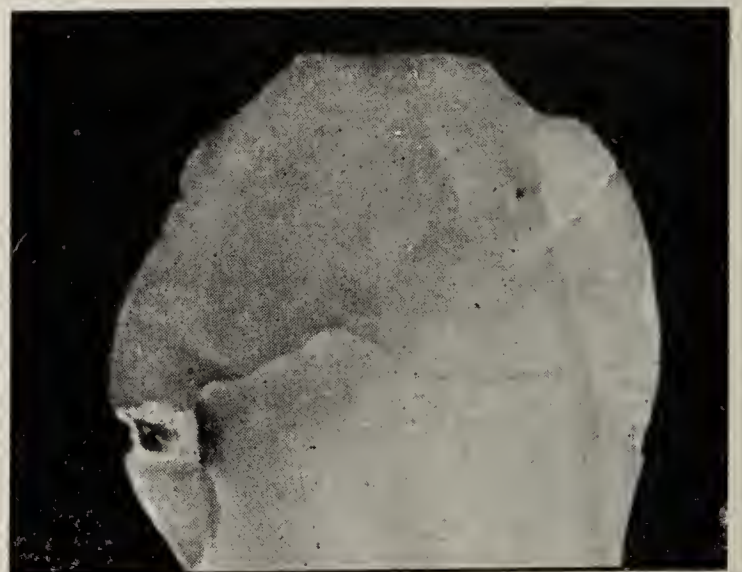


FIG. 180.

FIG. 179. A split lateral incisor, with a very white decay of enamel in its distal surface, showing a modification of the conical form of penetration. This has just penetrated the enamel, and the hyaline area, which may already be traced to the pulp chamber, is usually dark for such a case.

FIG. 180. A very narrow area of decay penetrating the enamel in the distal surface of a cuspid. In this the action of acid has been confined to a very narrow area of the surface of the enamel. The decay has reached the dentin, following accurately the length of the enamel rods. The enamel rods have fallen out of its central portion, microorganisms have been admitted and decay of the dentin has begun.

and that the central portion has just penetrated to the dento-enamel junction and is quite a little in advance of the general conical form of the invasion of the enamel. Decays occur, however, that show very little or none of this spreading upon the surface, even upon the proximal surfaces of the teeth, as will be seen from an examination of Figure 180. The illustration is more enlarged in order to show this spot to better advantage. Here it will be seen that a cavity has formed in the enamel. Some of the enamel rods have fallen out and the effect of the acid has passed entirely through the enamel and begun to dissolve the calcium salts from the dentin, and yet the area of the enamel affected, as seen in this dimension (the tooth having been cut mesio-distally from occlusal to gingival), is no larger than the shaft of a pin. The enamel rods have been exactly followed lengthwise, and the surface of the enamel



FIG. 181. A photomicrograph of a small area of the beginning of caries in enamel that shows an unusually smooth rounding of the deep line of penetration. D. Dentin. E. Enamel. The dento-enamel junction is between these two letters. X. A distinct swelling of the decayed area. This swelling is common in areas of decay in enamel.

about it seems free from the action of acid. Again it might be asked: By what power, circumstance or condition has the action of the acid been confined to this narrow area?

This following of the enamel rods is better seen in photomicrographs, in which the enlargement is only just sufficient for the

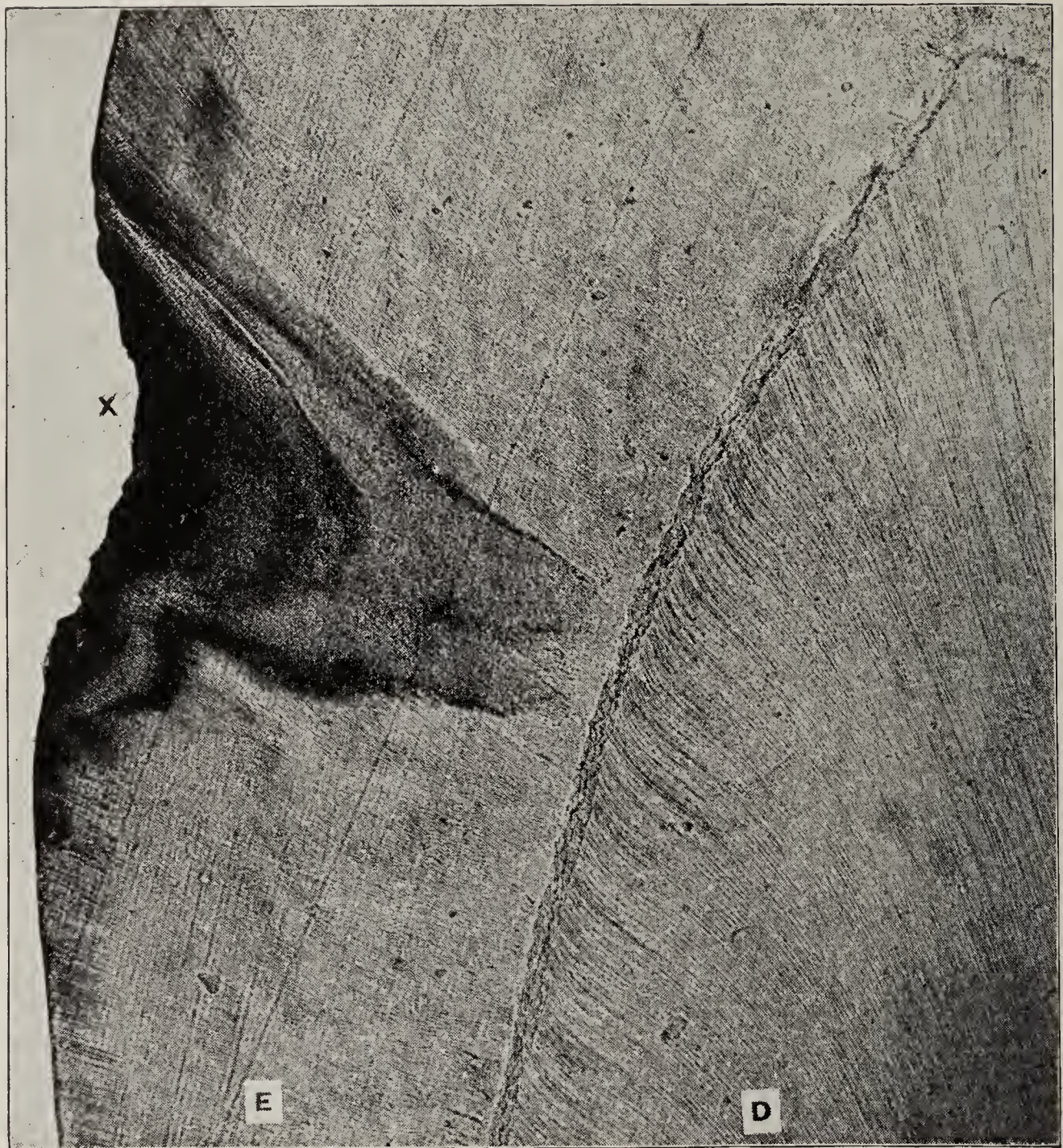


FIG. 182. Caries of enamel. D. Dentin. E. Enamel. X. Area of decay. In this figure the outer ends of the enamel rods have been broken away in grinding. Notice particularly the flame-like tongues of penetration sweeping toward the dento-enamel junction, following the lines of the length of the enamel rods. Also the penetration on the margins of the principal area are seen to follow the lines of the length of the enamel rods. This rigid following of the enamel rods is a characteristic of caries of enamel beginning in smooth surfaces.

direction of the enamel rods to be made out. It must be understood that a decayed area that is white to reflected light, shows an opacity to transmitted light and is therefore dark in the photomicrograph unless it is ground excessively thin. This group of sections, Figures 181, 182 and 183, were all cut from incisal to gingival. Beginning decays of the enamel cut in this direction show but little superficial spreading as compared with that seen in horizontal section, which will be shown later. The characters as to penetration, however, are the same, only less extended laterally.

Figure 181, a photomicrograph from a proximal decay, is almost unique in the smooth roundness of its deeper portion. The swelling of the decayed area is well seen at x. This is constantly



FIG. 183. A broader area of carious enamel. D. Dentin. E. Enamel. X. Area of decay. Here also we find the same inclination to the formation of flamelike tongues sweeping toward the dento-enamel junction, following the length of the enamel rods. There is no spreading of decay across the length of the enamel rods. All spreading is in new beginnings to the side on the surface of the enamel.

seen in microscopic sections of decays at this stage when no enamel rods have been lost during the preparatory work.

Figure 182 is a photomicrograph from a proximal decay of the enamel at x, which is very narrow on the surface and penetrates almost to the dento-enamel junction at the point of the

cone. This cone is of somewhat irregular outline. In this case the outer ends of the enamel rods were lost in grinding. The tendency is for decay to advance in flamelike tongues or projections, each following the length of the enamel rods. This is characteristic of caries of enamel, and often, when examined in the very early beginning, the starting points are divided from each other with these flamelike tongues projecting inward toward the dento-enamel junction. This is seen also in Figure 183 in a decay at x, which is much broader and flatter, showing less of the conical form. In this, a nidus, or beginning point, upon the surface of the enamel has been just above the letter x, and it has spread quickly over the surface to the gingival and the incisal of this point by the increase in size of the growing colony of microorganisms on the surface. Each new beginning, with individual enamel rods, or groups of rods, follows exactly along its length toward the dento-enamel junction. Other illustrations will show this tendency more prominently than these two.

PENETRATION OF THE ENAMEL IN BUCCAL AND LABIAL SURFACES.

Caries beginning in the buccal and labial surfaces presents the same characteristics as to penetration of enamel and spreading in definite directions as decays beginning in the proximal surfaces. In this case the line of beginning is along the margin of the gum in the middle third of the surface mesio-distally. The lines of extension in spreading are toward the mesial and the distal angles of the tooth. The beginning of these decays often forms a narrow whitened line, the length of which is from mesial to distal. These, taken with the decays of the proximal surfaces, tend to form lines encircling the teeth along the free margins of the gum, excepting that in neither case does the decay extend to the angles of the teeth, as previously mentioned in discussing proximal decays. Frequently, in the first beginning of the decay on buccal surfaces, there will be several starting points which later run together, forming a continuous line of whitened enamel. This gives, in cross sections of the tooth, appearances almost exactly similar to those shown in Figures 165-169 inclusive. The rule is that the extension of decay on the surface of the enamel is stopped by the friction of food in mastication before it reaches the mesial and distal angles of the surface. *Therefore, in the preparation of buccal and labial cavities in gingival third positions, mesial and distal margins should be placed beyond the area of liability to surface extensions of decay and within the areas of immunity, toward, or near to, the angles of the teeth.*

This subject will be presented in greater detail under the consideration of the clinical features of caries.

CARIES OF DENTIN

ILLUSTRATIONS: FIGURES 184-193.

Caries of the dentin can not occur until after the enamel has been penetrated. The enamel has no natural openings into which microorganisms can grow; and these have no power of penetrating into it, except as it is dissolved and removed by the acids which they form during their growth. Therefore, in decay of the enamel, the microorganisms producing the acid are on its surface. On the other hand, the dentin is everywhere permeated by canals into which microorganisms may grow when the dentin is exposed by the destruction of the enamel.

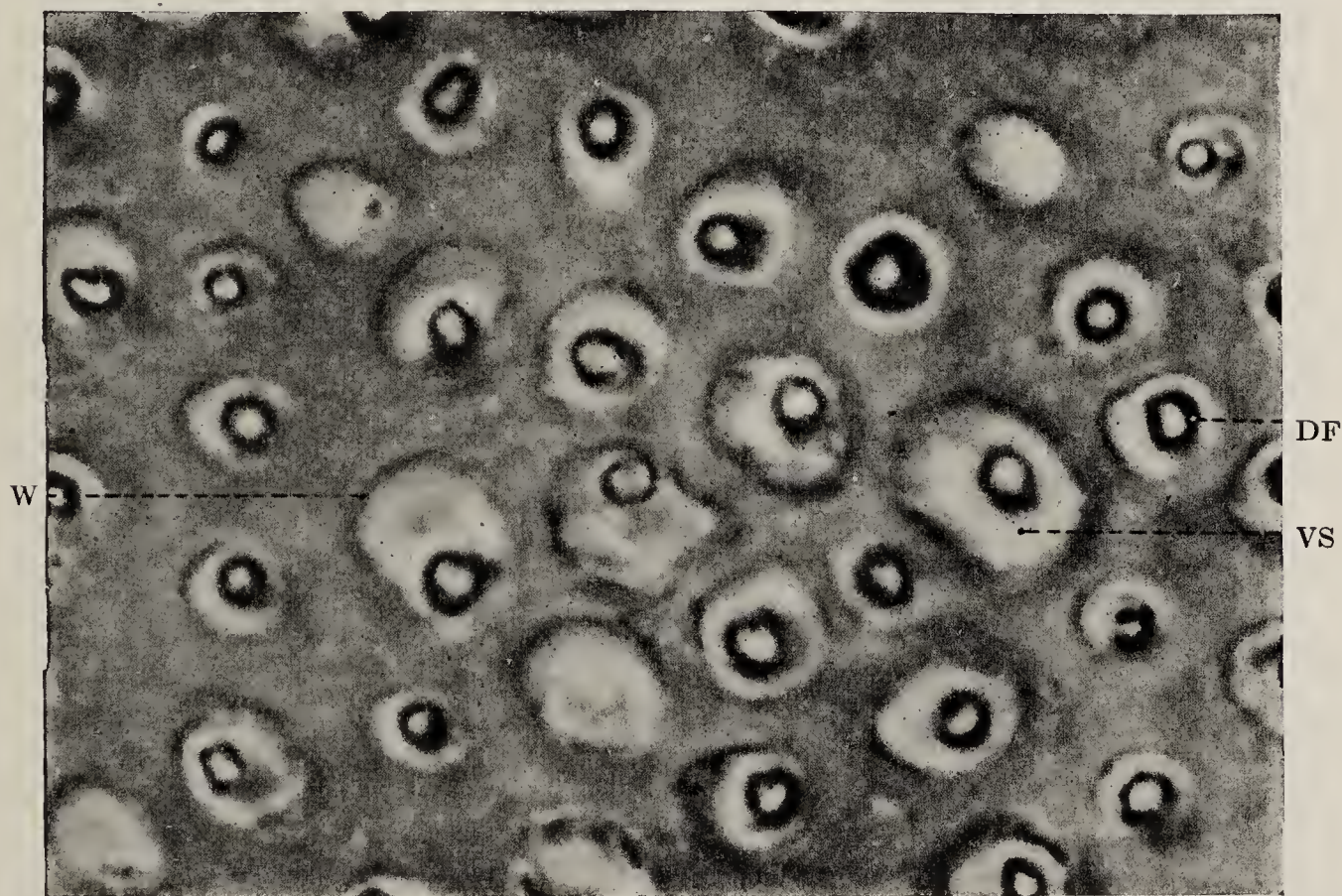


FIG. 184. Cross section of the dentin, showing the dentinal tubuli with (dentinal) fibrils *in situ*. Hematoxylin-eosin staining Magn. $\pm 4000\times$. W, wall of tubule; VS vacant space produced by shrinkage of dentinal fibril. DF, dentinal fibril. Some tubules are empty. (Meyer's Histology and Histogenesis. Churchill.)

Figure 184 is a section made across the direction of the dentinal tubules, and shows the dentinal fibrils. The white space surrounding each fibril was produced by the shrinkage of the fibril in preparing the specimen. Figure 185 is a higher magnification of a section cut parallel to the length of the tubules which shows a single fibril within a dentinal tubule.

Caries of dentin is caused by the same agency as caries of enamel; namely, the growth of acid forming microorganisms. In

caries of dentin, the microorganisms grow into the dentinal tubules and form their acid product within the tissue itself. This dissolves the calcium salts of the dentin, converting it into the soluble salt, calcium lactate, which gradually escapes into the surrounding saliva by osmosis. If there is a solution of sugar in the saliva and a solution of calcium lactate in the depths of a carious cavity, that part of the matrix of the dentin which is already softened by the solution of its calcium salts, acts as a dialyzing membrane, passing the sugar in and calcium lactate out. In this way, the growing microorganisms receive sugar from the saliva, and lactic acid (their waste product) which has become calcium lactate by combination with the calcium salts of the tooth, is eliminated. This process

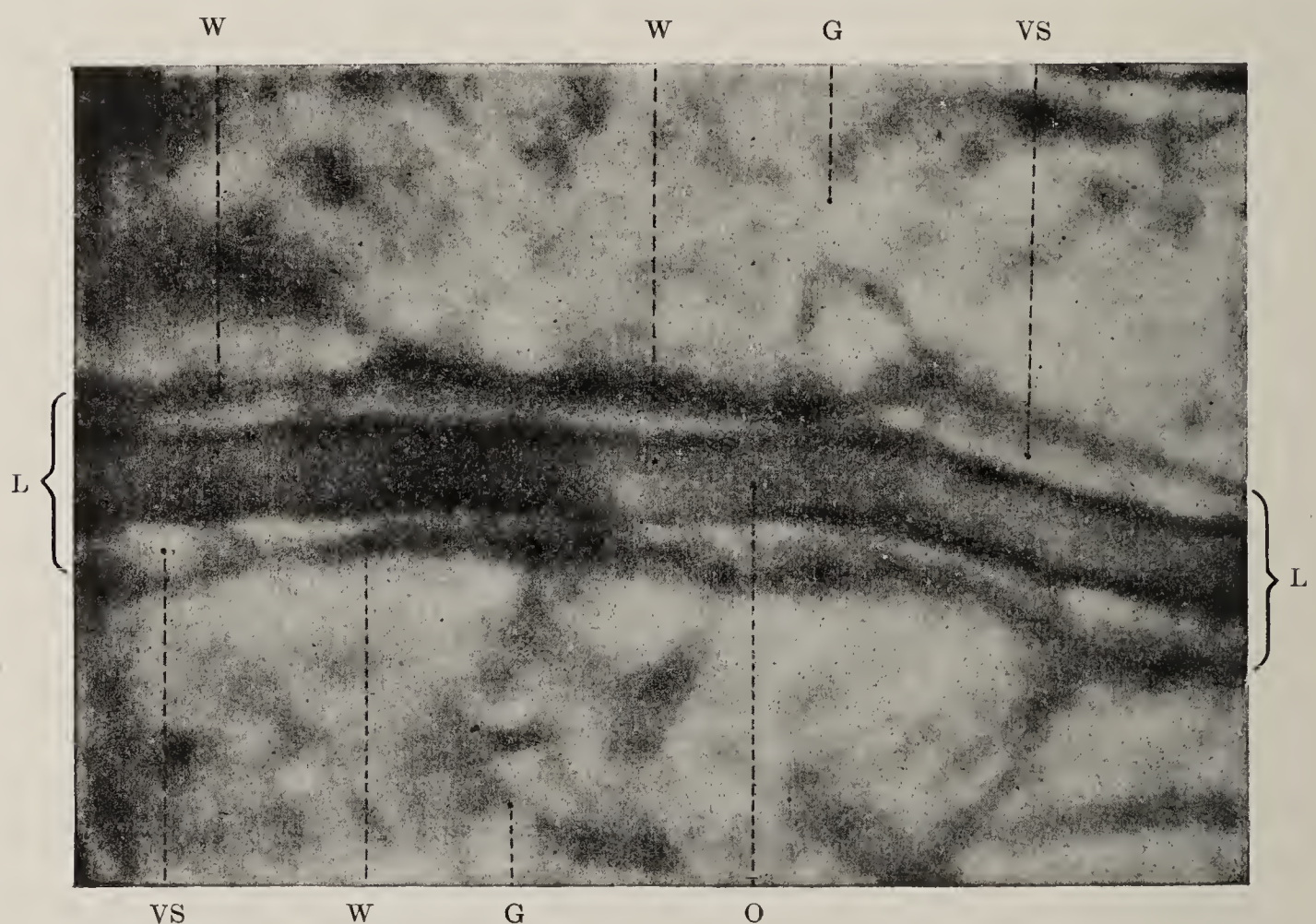


FIG. 185. Dentinal tubule with cytoplasmic extension of an odontoblast *in situ*. Hematoxylin staining. Magn. $\pm 5000\times$. W, wall of tubule; G, ground substance; VS, vacant space produced by shrinkage of dentinal fibril; O, odontoblastic extension; L, lumen of dentinal tubules. (Meyer's Histology and Histogenesis. Churchill.)

naturally goes on very slowly, so that weeks and months are required for any considerable cavity to form, and often several years.

In penetrating into dentin, microorganisms follow the dentinal tubules, simply growing into them as a grapevine would grow through a lattice. The anastomosing loops from tubule to tubule, which are plentiful near the dento-enamel junction, and any other openings such as interglobular spaces which they may encounter, are filled full as they go. There is much difference in the number and size of the anastomosing loops from tubule to tubule in differ-

ent teeth. In some these are very plentiful, in others very limited. In all, however, there is a sufficient number of these near the dento-enamel junction to afford a moderately free passage of microorganisms from one tubule to another. In a considerable proportion of teeth there are many small interglobular spaces near the dento-enamel junction, known as the "granular layer of Tomes," through which microorganisms may readily grow. Therefore among different teeth there are differences in the facility with which microorganisms will spread along the dento-enamel junction. The organisms having gained access to the dentin by the solution of the enamel, they grow into the dentinal tubules directly toward the pulp. They are continually gaining access to other tubules by spreading laterally in all directions along the dento-enamel junction from the first point of penetration of the enamel. *Therefore, the tendency is to the formation of a conical area of decay with the point of the cone toward the pulp of the tooth and its base against the dento-enamel junction.*



FIG. 186. A bicuspid split mesio-distally through an area of decay and photographed before separating the halves. When these are opened like a book, the penetration of the decay will be disclosed. These cuts have been made for the illustrations in an especially arranged lathe, in which the tooth is mounted on a slide rest.

The breadth of the cavity thus formed, in relation to its depth, will naturally depend upon the comparative rapidity with which the organisms may spread laterally along the dento-enamel junction. For this reason some cavities are broad and some are very narrow as compared to their depth.

In the illustrations of this subject many "split teeth" will be used. In these the teeth are cut through the decayed area as shown in Figure 186. The cut surfaces are polished and the parts laid open like a book and photographed as opaque objects. The half-tone engravings are made from the photographs without any retouching whatever. In many cases only one of the halves has been used.

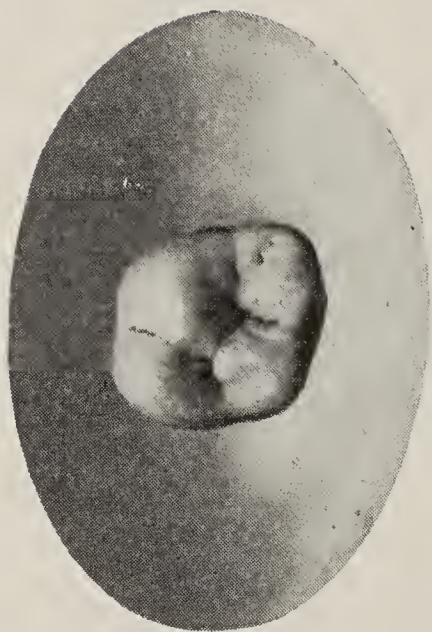


FIG. 187A.



FIG. 187B.



FIG. 187C.

FIG. 187A. The occlusal surface of an upper first molar with a decay in the central pit. This tooth was split mesio-distally and is shown in Figure 186.

FIGS. 187B, 187C. Other upper first molars showing, apparently, similar decays.

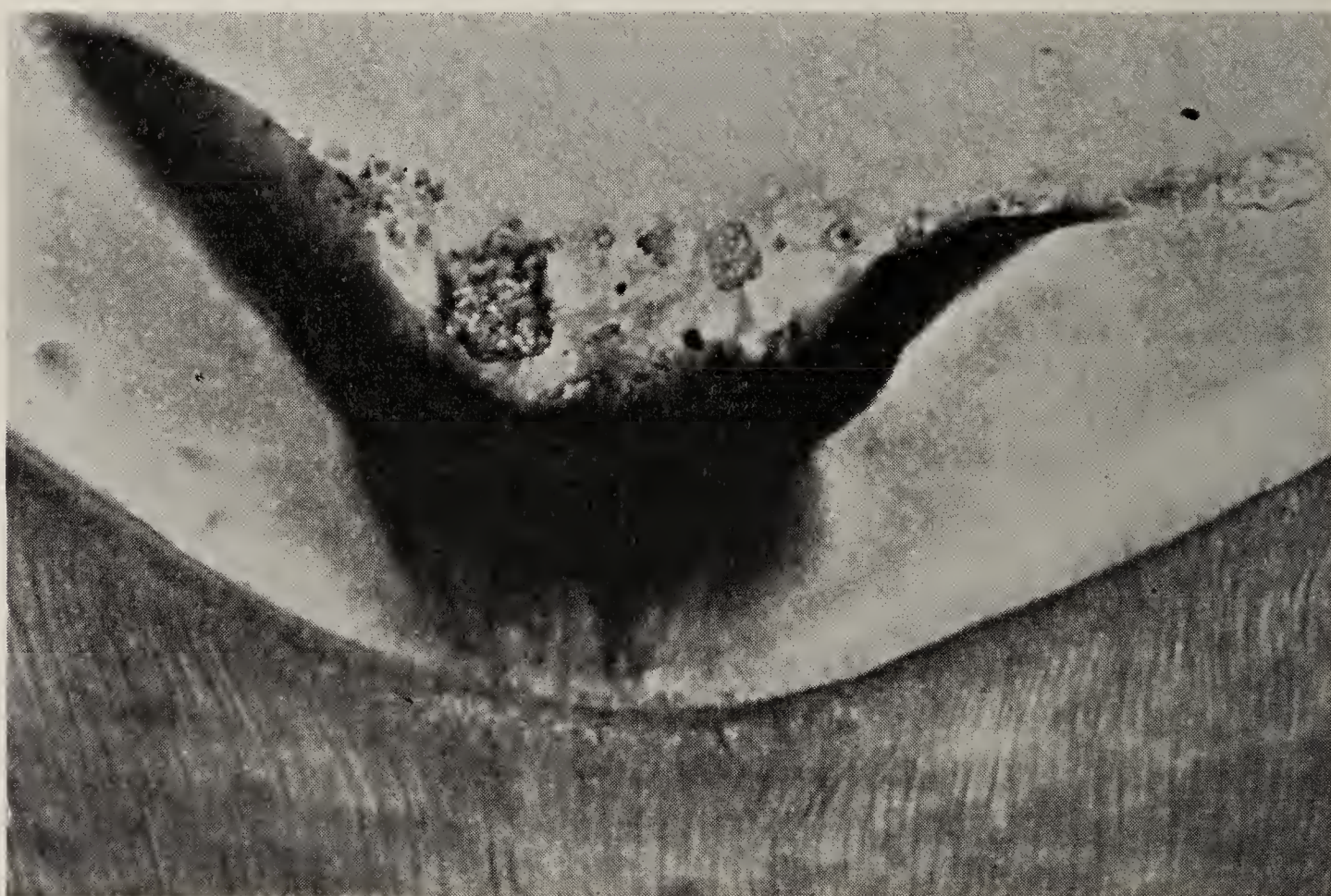


FIG. 188. High magnification of an occlusal decay in which the acid has very slightly penetrated the dentin.

The form taken by decay in dentin when it has begun in an occlusal surface is well shown in Figure 189, a photograph from a first molar split in half mesio-distally through the central pit, in which the decay began. The occlusal surface of the tooth before cutting is shown in Figure 187A. Other photographs of molars showing decays which, to superficial observation, seem to be similar, are shown in Figures 187B and 187C.

Figure 188 shows an occlusal decay which has just penetrated the enamel and the acid has invaded the dentin very slightly. In Figure 189 the wide spreading of caries along the dento-enamel junction forming the base of the cone, and the point of the cone reaching to the pulp chamber are well shown. This is the most common form. In this case, as in most cases of split teeth displaying caries, the cut surface is photographed as an opaque object.



FIG. 189. The upper first molar, Figure 187A, split mesio-distally, the surfaces polished, laid open and photographed to show the penetration of decay. The specimen shows particularly well the typical conical form of the penetration of dentin as it occurs when the opening in the enamel remains small. It also shows well the spreading of decay along the dento-enamel junction of the occlusal surface forming the broad base of the cone of the area of decay.

In examining such illustrations, it must be remembered that a section through a cone gives a triangular figure. Figure 158 is a good illustration of the spreading of decay along the dento-enamel junction in a temporary molar.

Figure 190, a lower second molar that has been cut buccolingually, shows a decay that has begun in the buccal pit. This decay has proceeded very slowly. There is wide burrowing along the dento-enamel junction related above, and the effect of the continued irritation during the slow progress of the decay is seen in the reduction of the size of the pulp chamber by the deposit of secondary dentin. This condition, resulting from long continued irritation of the dentinal fibrils, is a common effect. It occurs also in abrasion and erosion.

Careful observation has shown that microorganisms do not begin to grow into the dentinal tubules until the calcium salts have been dissolved out for some little distance in advance of them.

In the natural order of the advance of decay this is accomplished in the beginning by the infiltration of acid through the enamel before the enamel rods have fallen away, so as to admit microorganisms to the dentin. Therefore, when they are admitted to the dentin, some portion of it has been softened and the organisms begin growing into the tubules at once. As they do so the acid which they are forming infiltrates into the dentin in advance, dissolving more and more of the calcium salts. In this way it happens that the dentin is continually softened in advance of the growing organisms so that there is a little space softened around them that contains no microorganisms.

Figure 191 shows a few dentinal tubules which contain microorganisms. As the organisms fill the dentinal tubules, the tendency

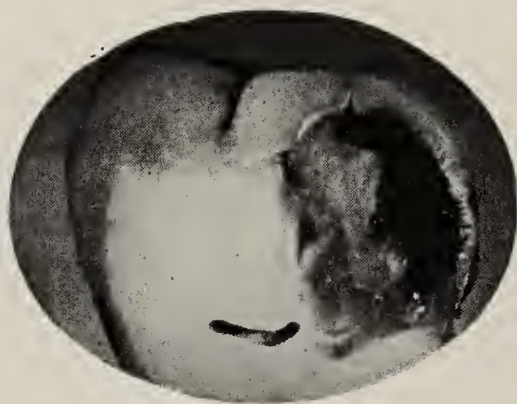


FIG. 190. A lower second molar with a large area of decay which has begun in the buccal pit. The decay has spread along the dento-enamel junction, undermining the greater part of the enamel of the buccal half of the tooth and has destroyed nearly one-half of the dentin. This injury has occurred while the opening into the cavity has remained small.

is to become more and more crowded together and the tubules begin to be enlarged. In some cases this enlargement is a very regular increase in size along the length of the tubules, the outer ends of which are enlarged most, as shown in Figure 192. In other cases there is much tendency to irregular swellings of the tubules, as shown in Figure 193. Indeed, these two illustrations have been chosen as showing the extremes of regularity and of irregularity in this respect. Every variation between the two may be found. Also the number of side branches into which organisms may grow varies indefinitely; after passing a little way from the dento-enamel junction some cases show very few or even none at all, while in others they are very plentiful.

This enlargement of the dentinal tubules continues until the division walls disappear, uniting two in one, three into one, and so on until there is nothing left but a mass of microorganisms mingled with some undissolved shreds of organic matrix, which, if the cavity is exposed to the saliva, wastes out and is washed away.

The acid producing organisms are facultative anaërobic. Within the author's observation, caries of dentin has seemed to progress most rapidly when closely shut in by overlapping enamel, and less

rapidly when the opening to the fluids of the mouth was broad and ample. In case the opening of the cavity becomes very broad while the cavity is yet shallow, the progress of decay is apt to be much slower. Finally, when the carious area is so flat as to be kept clean by mastication and is fully exposed to washings by the saliva, the decay ceases. The explanation for this difference is that when the opening to the cavity is very broad, much of the acid formed by the microorganisms is washed away by the saliva. This must be con-



FIG. 191. Microorganisms in dentinal tubules. The dento-enamel junction is at the top in the illustration.

sidered a local factor. The intensity of the condition of susceptibility, which will be discussed later, must also be reckoned with as a general factor when considering the rapidity of caries.

The progress of caries is limited, or even stopped, in a number of different ways. The crowding of meats into a cavity and the establishment of putrefactive decomposition, an occasional occurrence, is apt to end the progress of decay for the time, and leave the cavity with smooth, hard, blackened walls by the decomposition of all of the organic matrix from which the calcium salts have been removed. Decay may rebegin in this if conditions are so changed as to favor it.

When most of the crown of a tooth breaks away, the progress of caries will necessarily be across the length of the dentinal tubules, because these become horizontal on a level with the pulp of the tooth. If the organisms are prevented from entering the

tubules from the pulp canals, the progress of decay will be very slow, or there will be no progress at all.

When the pulp of the tooth has died and alveolar abscess in the chronic form is established with a free discharge through the root canals, the progress of decay is generally stopped as long as this condition continues. If, however, the apical portion of the root

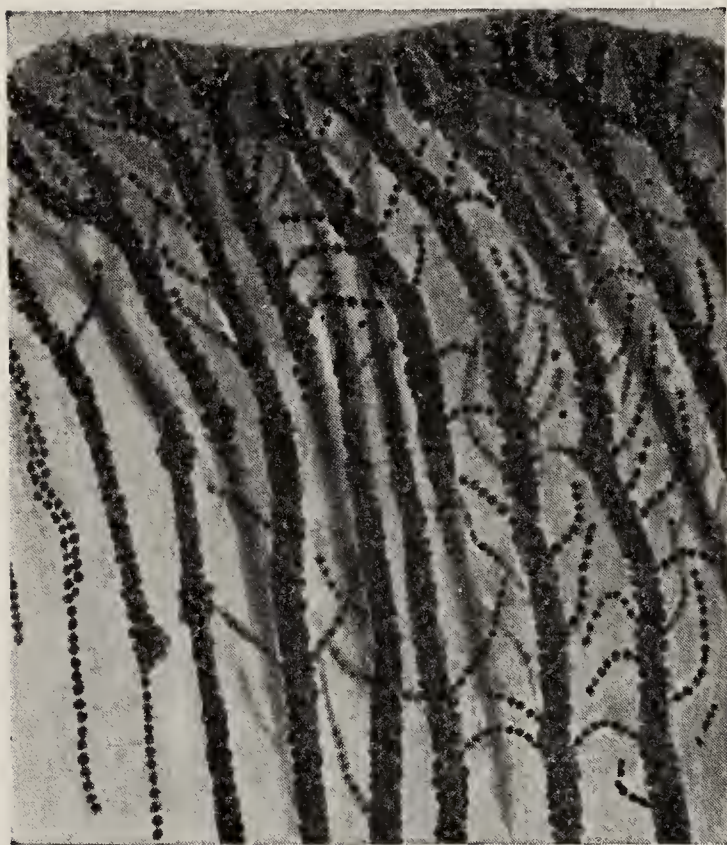


FIG. 192.



FIG. 193.

FIG. 192. An illustration showing the filling of the dentinal tubules with microorganisms. The dento-enamel junction is at the top of the illustration. The organisms have entered the tubules and by continued growth and multiplication have filled and enlarged them very evenly. Only a few slight local swellings of tubules are seen.

FIG. 193. Another illustration of the filling of the dentinal tubules with microorganisms, in which, as compared with Figure 192, the opposite extreme as to side branches and irregular swellings of the tubules is shown. This figure represents something like a maximum of irregular swellings of tubules and with no side branches. This is taken from deeper in the tooth close on the deeper margin of the invasion. The two specimens were from different teeth, and have been selected as representing the extremes of smoothness of the filling and enlargement of the tubules and maximum of filled side branches, as seen in Figure 192; and absence of filled side branches and something near the maximum of irregular swellings of tubules, as seen in Figure 193.

canal be choked by debris or otherwise closed, decay may proceed from the root canal, penetrate the tubules and rapidly hollow out the root to a conical shell and destroy it.

The breaking away of the lingual or buccal wall of proximal cavities is often a factor in saving a tooth from destruction, especially among those people who live much on coarse food. This has been found in the examination of some of the older Indian remains, especially of those tribes that were supposed to eat much parched corn. The food forced through the cavity in mastication and out through the broken side kept the surface worn smooth. The opportunity to know and to watch this among our own people occurs frequently.

The question as to whether caries is produced by a single species of microörganism or whether a number of kinds are acting together is often asked. As to this, there are several organisms found in the saliva of practically every person that have physiological characters which seem to fit them for the production of caries, and no reason is known why they may not be acting together in the same carious cavity. Dr. Miller seems to have found them so. In the deepest portion of the carious area the author usually found but a single variety, a streptococcus. In the decaying mass, however, pretty much all of the varieties found growing in the mouth may be found and some of them penetrate deeply into the softened portion. Especially a white staphylococcus is often found deep in the dentinal tubules, if judged by the difficulty of keeping clear of it in the effort to get a pure culture of the streptococcus from carious dentin.

CLINICAL FEATURES OF CARIES

ILLUSTRATIONS: FIGURES 187-229.

Particular attention is called to the diagnosis of dental caries and the various methods employed in discovering beginning decays, in the chapter on Examinations of the Mouth, page 31.

In the further consideration of the injuries inflicted by caries of the teeth, caries of both the enamel and dentin may be included. The processes may be discussed as a whole, noticing the various characters presented in the beginning and progress, controlled purely by local or clinical conditions. These conditions have no special signification as to immediate or remote cause of caries further than location or condition of tissue which may influence the action of these causes. Yet these causes which are brought into action, seemingly because favorable conditions for their active development have been presented, can not be lost sight of in any consideration whatever. It may truly be said that, *without the presence of the principal causes no decay could occur, and also that, without reasonably favorable conditions for their action, these causes would not produce decay.*

This may be said to constitute the clinical features of dental caries, and in its consideration frequent reference will be made to the clinical management of cases. One of the surprising features of the study of dental caries that comes sharply in view when the history of the development of our knowledge of it is closely scanned, has been its divorcement from all clinical consideration. It seems to be correct to say that a large number of practitioners of dentistry are making restorations, without proper thought of the relations which their plan of treatment may bear to the conditions that have localized the decay being treated at that particular spot; also without making adequate provision, or too often making no provision at all, for the prevention of a recurrence of decay.

Studies of the conditions of the beginning of caries of the enamel are all-important in the study of dental caries, considered from the clinical standpoint. One of the noblest pieces of scientific work in pathology was Dr. W. D. Miller's investigation of dental caries. But this investigation was confined to the immediate active cause of caries as it occurs in dentin. Decay of enamel, or the conditions localizing beginnings of this, which constitutes the initial lesion, without which caries of dentin never occurs, must be fully understood for the effective application of preventive measures. These constitute the principal clinical features of the disease under consideration. Knowledge of them is, as yet, but

partially developed, particularly in that the systemic conditions of susceptibility and immunity are but indefinitely known; and observers do not yet agree as to the especial relations of saliva and of microorganisms to the particular processes in the earlier parts of the beginning of caries of enamel.

THE TEMPORARY TEETH.

In most respects the clinical features of caries of the temporary teeth are the same as for the permanent teeth. Possibly the most important difference is in the early exposure of pulps. The thickness of both the enamel and dentin is less and the pulp chambers are larger in comparison with the permanent teeth. Therefore the period from the beginning of decay of the enamel to the exposure of the pulp is often very short — often a few months. This indicates the necessity of more frequent examinations of young children than of adults.

Maintenance of conditions of the temporary teeth which will permit vigorous mastication is of great importance in promoting the highest degree of immunity for the permanent teeth. If a child has a tooth that is painful when used in chewing, or if molar contacts are not maintained and the interproximal gum tissues become inflamed, food will be swallowed without mastication and the most important natural protection against caries is lost. This loss of vigorous mastication involves the first permanent molars, and many are hopelessly decayed before the bicuspid erupt.

The practice of extension for prevention need not be carried to the same limits in its application to the temporary teeth, depending upon the degree of susceptibility and the age at which the restoration is made. The life expectancy of the tooth is the guiding factor, rather than the life expectancy of the patient, as is the case with the permanent teeth.

The gradual separation of the temporary incisor teeth makes unnecessary the consideration of extension of cavity margins for prevention of decay, as practiced for the permanent teeth.

OCCLUSAL SURFACE DECAYS IN MOLARS.

ILLUSTRATIONS: FIGURES 187-189; 194-196.

PRINCIPAL CLINICAL FEATURES: (1.) Beginning in pits, absence of superficial spreading. (2.) Rapid lateral decay along the dento-enamel junction. (3.) Spreading in the dentin in true conical form. (4.) Great softening of dentin in advance of the decomposition of the organic matrix. (5.) Backward decay of enamel. (6.) Very large decays often occurring while the opening to the surface remains small. These characters are common to decays beginning in pits and fissures wherever found.

The conical form of decays of dentin beginning in pits, in the occlusal surface or elsewhere, the spreading along the dento-

enamel junction, and the absence of spreading superficially on the surface of the enamel, have been considered in previous articles and some principal illustrations have already been shown. For the further illustration of the clinical features, the Figures 194-196, inclusive, are here provided. It will be seen that Figure 194 and other figures of this group show the same form of decayed area more or less perfectly, the detail being different in some degree on account of size of the decayed area or the particular form of the surface of the tooth about it. The case illustrated in Figure 194, considering the tooth as a whole, is complicated by two other decays, both of them in the distal surface; one a very shallow decay, beginning in the enamel at the usual point of beginning decays, and the other a decay beginning in the cementum and penetrating into the dentin at the cemental line. This latter is a serious



FIG. 194. Photograph of a split upper first molar with an occlusal decay of ordinary form. Note a small beginning of decay of enamel in the distal surface, and a decay beginning in the cementum. This latter is characteristic of decays which occur because of crowding of food into the interproximal space, with resulting absorption of the interproximal gum tissue.

complication in the clinical sense, which has arisen from neglect of the leakage of food into the interproximal space. In Figures 195, 196, the openings through the pits, in which the decay has begun, are unusually large. This has occurred by the breaking away of the enamel about the pit much earlier in the progress of the decay than usual. In response to this early widening of the pit, giving a greater opportunity for washing out acids formed in the dentin, the penetration of the dentin presents a much less pointed cone, the penetration is less in proportion to the breadth than in cases in which there is less breaking of enamel about the pit. This is seen most distinctly in Figure 196. In studying these illustrations, one must divide the area of actual decay from the

cloud (hyaline area) stretching away toward the pulp. In Figure 195 there is really very little decay of dentin. The triangular (in section) cloud stretching toward the pulp chamber is not softened dentin. In Figure 196 the area of decay is more definitely outlined by the distinctly darkened area which is flattened or rounded rather than in the typical conical form. This is characteristic of a wide opening through the enamel. The rate of progress of decay is quite apt to be greater where it is hidden away under the overlapping enamel than in the central parts. The tendency, therefore, is to form broad, flat cavities. Both of these cases, considering each tooth as a whole, are complicated by proximal decays. On account of ease of access and the absence of the tendency to superficial spreading of decay, these cavities and pit cavities, wherever they occur, are the simplest of cavities in the clinical sense. The only thing requiring special attention outside of the area of decay and its full exposure by trimming away the undermined enamel, is to see to it that all fissures are cut out to such a point as will give opportunity for a smooth finish of the restoration.



FIG. 195.



FIG. 196.

FIGS. 195, 196. Photographs showing decays in occlusal pits, in which the opening through the enamel has become larger and the decayed areas are of less depth in proportion to their breadth. Some proximal decays are also shown.

PROXIMAL SURFACE DECAYS IN MOLARS.

ILLUSTRATIONS: FIGURES 197-200.

PRINCIPAL CLINICAL FEATURES: (1.) The beginning decays of this class are hidden from view in the proximal surfaces of the teeth, making their early discovery difficult. (2.) A tendency to wide spreading on the surface of the enamel, bucco-lingually, making extension for prevention necessary in restorative operations. (3.) Tendency to the early undermining of the marginal ridge by the extension of decay along the dento-enamel junction and the exposure of the cavity by its breakage. (4.) Wide secondary spreading gingivally in a considerable number of cases which is

liable to be overlooked in the preparation of cavities. (5.) Recurrence of decay at the bucco-gingival and linguo-gingival angles of restorations when judicious extension for its prevention is neglected. (6.) Recurrence at the gingival margins of restorations where contacts are of bad form, allowing food to leak into the interproximal space. (7.) Hence the requirement that correct forms be given contact points in the finishing of restorations.

For the discovery of decays in the proximal surfaces of bicuspids and molars see page 32.

The conical form of cavities in the dentin is a little different in proximal cavities, where seen in sections cut mesio-distally, from those in occlusal surfaces, because of the difference in the trend of the dentinal tubules from the line of the dento-enamel junction toward the pulp of the tooth. It is still a cone, however, with its base placed diagonal to its length, or in section it is a triangle,



FIG. 197. A photograph giving an excellent showing of a mesial and a distal decay in a lower molar that have made but little advancement. The two halves of the tooth are shown, giving two views of each decay. In the decay in the mesial surface the enamel rods have not yet fallen out from any part. The enamel has been penetrated, however, and in the half on the left side some solution of the dentin is apparent. But for the area of decay to occupy the contact point as in this case is infrequent. In the decay in the distal surface, the enamel rods have fallen out of the central area, and in the half on the right side there is an excellent showing of the projection of decay along the dento-enamel junction. The illustration is also an interesting one because of the showing of the unusual nearness of the pulp to the occlusal surface and the danger of exposing the mesial marginal ridge of the pulp, or horn of the pulp.

having one of its basal angles obtuse. The tendency to this form is best displayed in the decays in Figure 197. In general, there is a greater tendency to rounding of the line of invasion of dentin than is seen in the decays beginning in occlusal surfaces. There is the same tendency to wide softening of the dentin more rapidly than the decomposition of the organic matrix when the opening in the enamel is small, as is seen elsewhere; but this disappears when the enamel breaks away, exposing the cavity to the occlusal surface. As the time in relation to the progress of the decay at which this breakage of the enamel occurs is very variable, extensive burrowing with large amounts of softened dentin is liable to be found unexpectedly in that which seemed, upon superficial examination, to be a small cavity. The large proximal decays in Figures 199 and 200 show something of the extent to which these

cavities may burrow before the marginal ridge breaks away, exposing them to the occlusal surface.

A lower molar is shown, in Figure 197, with a mesial surface decay and a distal surface decay, both of which are excellent types of the early beginnings of caries in these surfaces. In the mesial surface, the decay has penetrated the full depth of the enamel, no enamel rods having yet fallen away. In the distal surface the enamel rods have fallen out and the extension of caries along the dento-enamel junction is making progress. This is seen best in the picture to the right. From this the hyaline zone stretches away to the pulp chamber. This picture is a most excellent study. It is well to note the small amount of dentin between the occlusal surface and the pulp in this case, and also the great extension of the mesial marginal ridge of the pulp. The frequent extension of the mesio-buccal horn of the pulp in both upper and lower first molars is a menace in cavity preparation that should be carefully guarded against when possible, by avoiding cutting through



FIG. 198.



FIG. 199.

FIG. 198. An upper first molar with a distal decay which has made considerable progress, and has undermined the distal marginal ridge.

FIG. 199. The decay on the right side of the illustration shows a very prominent clouding extending to the pulp. This also shows particularly well the form of the clouded area, with the base of the cone placed diagonal to its length, caused by the relation of the direction of the dentinal tubules to the proximal surfaces. The actual decay of the dentin in this case is marked by the very dark area about the small opening in the enamel. The spreading along the dento-enamel junction is very wide. The decay in the mesial surface has exposed the pulp before the mesial marginal ridge has broken so as to expose the cavity.

its recessional line. This recessional line is usually nearly a direct line from the point of the cusp to the present location of the point of the horn of the pulp. Where the point of the horn may be located along that line in any individual case can not be told in advance, but that it will not be much out of that line is a certainty. When it is possible, cutting that line should be avoided.

In this group of pictures, and in the next following, the clouded areas stretching away from the areas of decay toward the pulp are particularly prominent. The best examples are in Figures 199 and 205. It is an almost constant appearance in some form, even in the freshest decays, after the acid has begun to dissolve the calcium salts of the dentin, but it never appears before

the enamel has been penetrated. It was first described by John Tomes and by him was called the hyaline area in advance of actual caries. It finally became known as the hyaline area of Tomes. At first Mr. Tomes supposed this was caused by an increased deposit of calcium salts, filling the dentinal tubules. While the chemico-vital theory of caries was held, this was looked upon as an effort to bar the further progress of caries by building against it. With further studies, this explanation became untenable. The appearance differs much in different cases. It may be either a cloud fringed with white, or a white area fringed with cloud, an irregular distribution of these, or it may be wholly of the one or the other. How much actual injury to the tooth is done by that influence which causes this hyaline zone can not be told. That there is a distinct injury is certainly true. It seems now that the most rational ex-

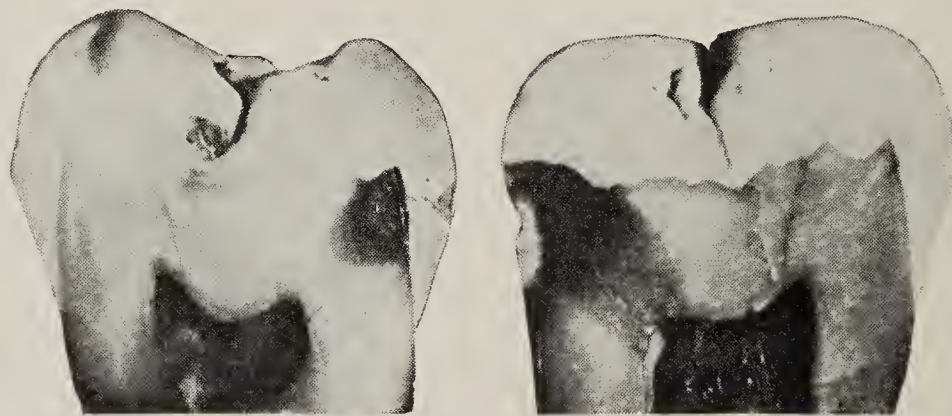


FIG. 200. This tooth has an unusually heavy enamel cap. This seems not to have been any bar to the penetration of caries, but has prevented the breaking of the marginal ridge disclosing the cavity to the occlusal surface, though it is extensively undermined.

planation is that in this zone many of the dentinal fibrils are degenerated from the irritation caused by the progress of caries. If this is true, the extension of this to the pulp may be the cause of the hyperæmia of the pulp that so often occurs in carious teeth before the pulp has become involved or exposed to the actual carious process.

In studying the decays of this tooth, Figure 197, or others of this group, it will be seen that any effort to prepare these smaller cavities as simple proximal cavities, would inevitably produce conditions which would cause failure of the restorations, for the reason that they are situated so close to the marginal ridges that the enamel rods slope very much toward the ridge. Further cutting in that direction to obtain a clean enamel wall, or to remove the decay projecting along the dento-enamel junction, would bring the margin of the cavity to an impossible position, due to the direction of the enamel rods. Therefore, the marginal ridge should be cut through and anchorage made in the form of a step in the occlusal surface.

This is more sharply illustrated in the undermining of the

distal marginal ridge in Figure 198, in which decay has made a little further progress. In such positions, this undermining is generally done very early in the progress of the decay and the marginal ridge breaks away, exposing the cavity correspondingly early. This generally gives the patient the first suggestion of the presence of a cavity. Often, also, the dentist overlooks these decays until this breakage reveals them. This may be fatal to the pulp of the tooth, especially in cases in which the decay has begun farther to the gingival, as in the decay on the left, in Figure 199, or in those cases in which the enamel cap is unusually strong, as in Figure 200. In the decay seen in the right of Figure 199, the undermining of the enamel seems extreme. This is often the case in those decays that have penetrated the enamel as a small opening without much superficial spreading, as is apparent in this case. Finally it is well to notice that the very heavy enamel cap shown in Figure 200 has been no bar to the invasion of caries, but that, on the other hand, has been rather a menace in that the failure to break away early has kept the cavity hidden and maintained conditions favorable to the rapid advance of caries of the dentin.

OCCLUSAL AND PROXIMAL SURFACE DECAYS IN BICUSPIDS.

ILLUSTRATIONS: FIGURES 201-206.

PRINCIPAL CLINICAL FEATURES: (1.) In pit and fissure decays, the danger of undermining the mesial and distal marginal ridges by extension of caries along the dento-enamel junction, involving also the enamel of the proximal surfaces. (2.) In proximal surface cavities the clinical features are the same as in proximal surfaces of the molars.

The positions in which pit decays occur in the bicuspid are well shown in Figure 201. The occurrence of these, independent of caries of the proximal surfaces, is not nearly as frequent as in the molars, yet a considerable number are met with. If these are treated before considerable progress has been made, they are very simple cases; but, as decay progresses, it quickly undermines the marginal ridge and is liable to weaken the enamel of the proximal surface to such an extent that this must be cut away to make a safe restoration. In the illustration, Figure 201, which presents decays of the enamel in each pit and in its distal surface, it will be noted that, as these progress in the dentin, they will quickly undermine the enamel of both the marginal ridge and the proximal surface, and the enamel of these parts will be weakened by backward decay. This undermining often makes a proximo-occlusal restoration necessary even though there may have been no proximal decay.

Many of the proximal decays in bicuspid teeth begin near the marginal ridges, as in the molars. This is illustrated in the beginning of decay of the enamel in the distal surface in Figure 201. These undermine the marginal ridges and disclose the cavities early in their progress. This is seen also in Figure 202 in the decay on the right side of the figure and is much more plainly seen in the photomicrograph of the same decayed area in Figure 204. The excellent photograph of the split bicuspid in Figure 205, shows the most usual points of beginning and direction of progress of these decays to great advantage. It will be noticed that in the cavity on

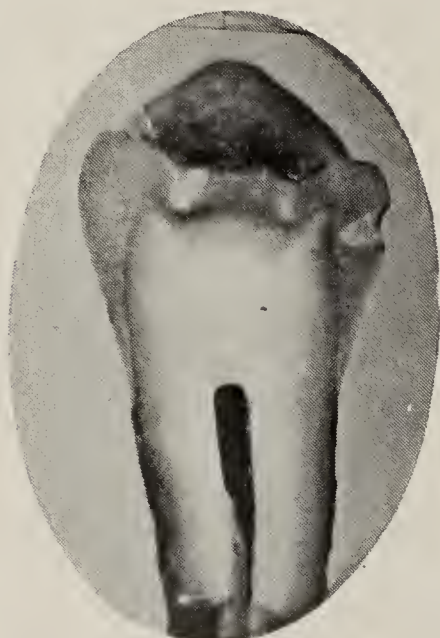


FIG. 201.

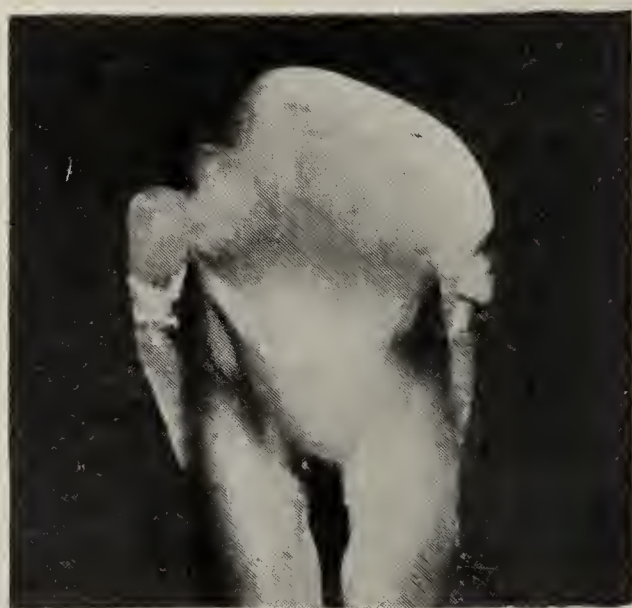


FIG. 202.

FIG. 201. A photograph of a split bicuspid disclosing three beginning decays which only just reach the dento-enamel junction. Any one of these will quickly undermine the dento-enamel junction if neglected. The pit decays, if allowed to become deep, often undermine the enamel of the proximal surfaces also.

FIG. 202. The hyaline area of the left decay is well shown. The extension occlusally of decay along the dento-enamel junction and the very white backward decay of the enamel are interesting features. After this photograph was made, the polished surface was cemented to a cover glass and ground thin for photomicrographing, and Figures 203, 204 were made.

the left side of Figure 205, decay has already begun to undermine the enamel forming the marginal ridge, and the distance to the pulp is so great that the breakage of the marginal ridge would probably occur, disclosing the presence of the cavity before the pulp would become involved. But this tooth shows that the pulp has receded and is smaller than usual. In many cases the pulp is involved before the breakage of the marginal ridge. This brings us to the necessity of discovering these decays at an early date in their progress in order to limit the injury to the dentin by caries and prevent the exposure of the pulp. In the split bicuspid, Figure 206, there is a mesial cavity which has extended in the dentin to the exposure of the pulp before the mesial marginal ridge is broken. This shows well the extension along the dento-enamel junction under the occlusal surface of the tooth. This great exten-



FIG. 203. Photomicrograph showing the carious area seen on the left of the small photograph, Figure 202. D. Dentin. E. Enamel. X. Area of decay. Y. Line of actual solution of the calcium salts of the dentin. Z. Backward decay of the enamel, which shows very white by reflected light, but is dark by transmitted light. In the drying of the specimen, the decayed dentin has shrunk and pulled a little away from the enamel. A slight break of the enamel rods has occurred at X, and a little contusion of the decayed rods has occurred near the letter Y. No enamel rods have fallen out, however, and microorganisms have not been admitted to the dentin.

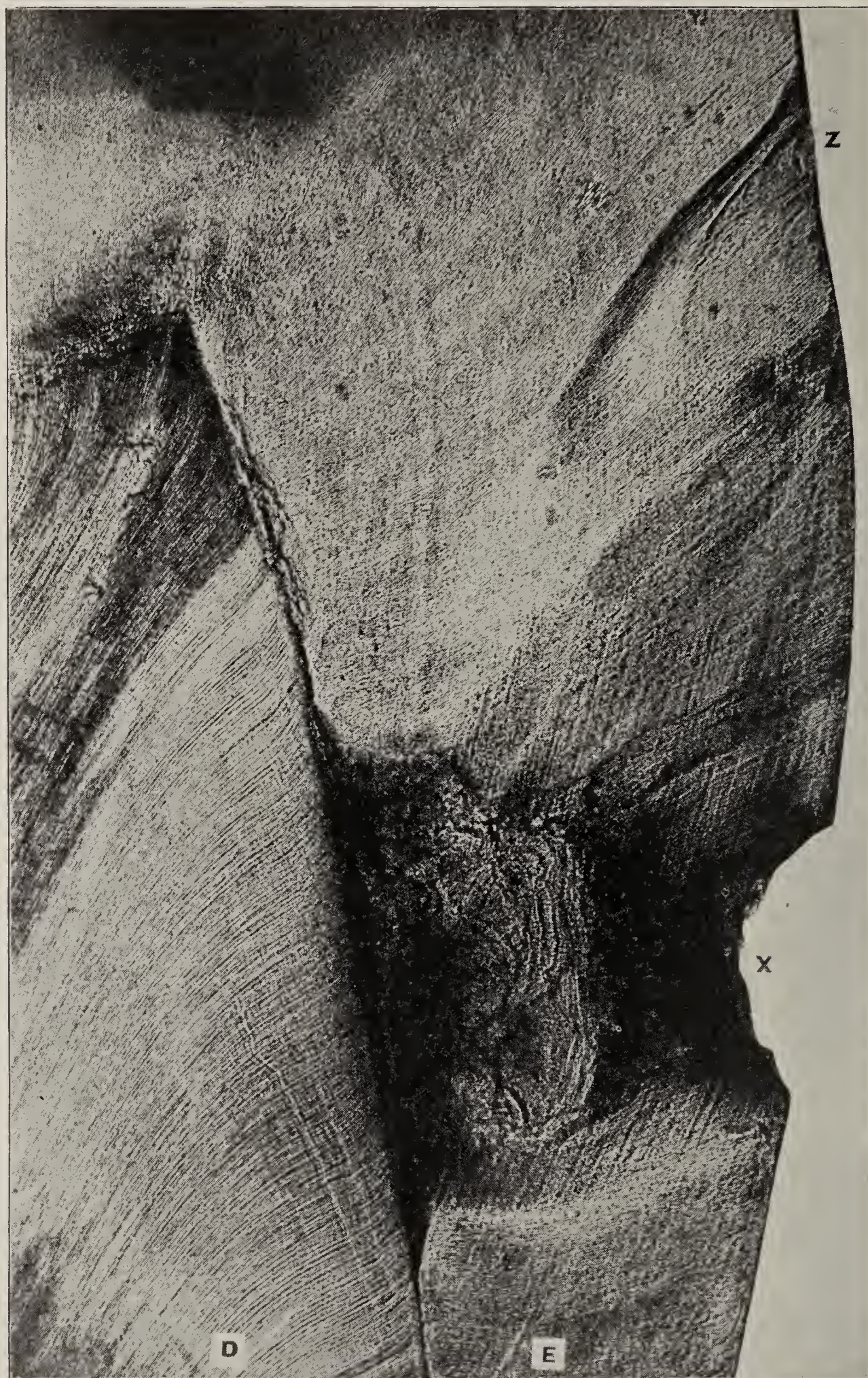


FIG. 204. Photomicrograph of the decayed area on the right side in Figure 202. D. Dentin. E. Enamel. X. Area of decay. Z. An extension of the superficial beginning of decay of the enamel occlusally. The dento-enamel junction is seen between D and E. In this case the enamel rods have fallen into a tangled mass in the deeper portion of the central part of the decay at X, leaving a partly open cavity in the enamel. The dentin beneath is considerably decayed but has not been pulled away from the enamel by shrinkage. The extension of the decay, as seen in the perpendicular section, has been toward the occlusal portion of the tooth. The extension of beginning on the surface seen at Z is in a degree separated from the principal area of decay and extends toward the dento-enamel junction in a flamelike tongue, following the course of the enamel rods.

sion along the dento-enamel junction and the general form of the cavity is typical of this class of cases in which the opening of the cavity remains closely covered by the proximating tooth.

Taken all together, the principal clinical differences between the proximal decays of the bicuspid and the molars are to be found in the smaller comparative size of the bicuspid in relation to the exposure of surface to the beginnings of decay. For this reason, the amount of sound tissue in proportion to carious tissue quickly becomes much less than in the molar teeth, and their successful treatment is for this reason rendered more difficult. These facts intensify the demand that closer examinations be made and



FIG. 205. This excellent photograph of a split bicuspid with mesial and distal decays is remarkably similar to the last, but in many ways a more perfect picture than Figure 202. In the decay on the left, the enamel rods are broken down and are lying in the cavity in the enamel in a tangled mass. In the decay on the right, the enamel rods are still in perfect position and no microorganisms have been admitted to the dentin. The dark portion of the dentin accurately exhibits actual decay of the dentin in both decays. The hyaline areas are both very well shown.

restoration resorted to earlier in the progress of caries in the bicuspid. If this will not allow the cutting to be made much narrower on the surface, it can be made much shallower, giving proportionally a much greater mass of healthy tissue to support restorations and to limit the danger of breakage.

The photograph from a split bicuspid, Figure 202, shows a cavity in the mesial surface, which is of especial interest. The form is fairly well outlined, showing particularly that the enamel rods have not fallen out. Yet the clouding of the dentin reaches to the pulp chamber. The acid, which has percolated through the decaying enamel, has begun dissolving away the calcium salts of the dentin. This extends along the dento-enamel junction, both

to the occlusal and to the gingival. In this picture the backward decay of the enamel, in the extension toward the occlusal, is particularly well shown. It is this backward decay of the enamel which so weakens it that it often breaks away early in the progress of the decay.

After the photograph of this tooth was made, the cut surface was cemented to a cover-glass, and this in turn to a grinding-disk, which was placed in the grinding machine, and a section ground thin enough for microscopic examination by transmitted light. From this, photomicrographs were made which show the carious areas in greater detail. The sides to which each belong have been preserved as they appear in the small photograph.

If the decay on the left in the photomicrograph, Figure 203, is studied, the amount of the solution of lime salts from the dentin,

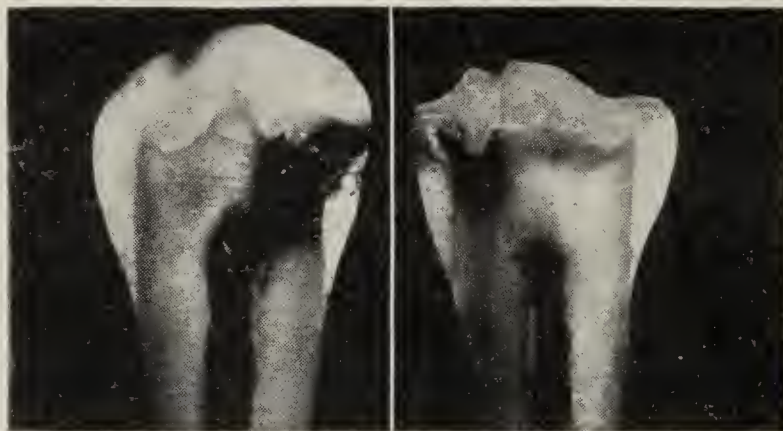


FIG. 206. The halves of a split bicuspid in which decay through an opening through the enamel that has remained small, has involved the pulp before the marginal ridge has broken, though it is extensively undermined.

as it is shown at Y, is easily followed. The injury to the dentin, however, extends from the point of the dentin cusp near the occlusal, down past the decay of the enamel toward the gingival. At Z the outline of the backward decay of the enamel, seen in the small photograph, is quite plainly shown, but by transmitted light it is dark. A backward decay toward the gingival is not so well shown, because of some little cracking of the enamel in that region, which mars the picture. The occlusal portion of this picture is upward, as it is in all of the photomicrographs.

The decay on the right of the small picture, Figure 202, is represented in Figure 204. Although this decay has not caused enough solution of calcium salts in the dentin to show shrinkage in drying, the injury to the dentin seems to be considerable. The enamel rods are broken down in the central area, which occurred in the process of grinding. It will be seen in the photomicrograph that many of the partially dissolved enamel rods lie in a tangled mass in the deeper parts of the cavity. The very unusual extension of the carious process in the enamel toward the occlusal at Z will also be noticed here, separated partially from the prin-

cipal area of decay, a flamelike tongue shoots inward from the surface and is making progress, following directly the length of the enamel rods. This represents a new decay of enamel in the form of an extension, but beginning upon the surface. It is not a lateral extension within the tissue, but marks the spreading of microorganisms on its surface. It is well to note particularly the direction of the enamel rods along the occlusal side of the flame-like tongue of decay shooting down from Z, with reference to the inclination of the enamel wall that would be required if this were prepared as a simple proximal cavity. It will be seen that this inclination of the enamel rods is too great to fill against safely, for it is in such a position that the margin of the restoration would not be sufficiently strong on account of its thinness.



FIG. 207.

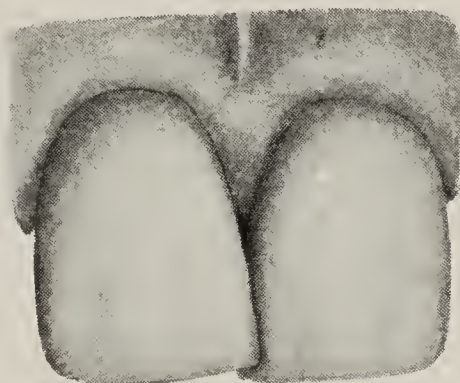


FIG. 208.

FIGS. 207, 208. Drawings showing an unusual form of decay in a central incisor in Figure 207, and the overlapping of the two central incisors in Figure 208, in such a way as to bring the surfaces of the teeth in very near approach in such a direction as to produce this form of beginning decay.

MISPLACEMENT OF BEGINNING PROXIMAL DECAYS.

ILLUSTRATIONS: FIGURES 207-208.

It has been noted, in considering Figure 166, that the proximal decay upon the mesial of the bicuspid is out of the ordinary position to the lingual, passing partially around the lingual angle of the tooth. Such displacements from the normal position occur frequently on account of irregularity of the teeth bringing their surfaces together in unusual relations to each other, or some such accidental condition.

In Figure 207 a peculiar shaped cavity is presented in the mesial surface of the central incisor, having a prolongation running labio-incisally. When the tooth is seen standing alone, such form of beginning decay might seem difficult to explain, but examination of Figure 208 explains the reason for this unusual form. The relative position of the two teeth is such as to bring very near contact directly along the line which this decay has taken in the enamel, and is the local influence which has caused this peculiarity. In any case, if a tooth, a bicuspid for instance, is turned one quarter around upon its axis, so that the buccal surface, proper, becomes

the mesial surface, and the lingual surface, proper, becomes the distal surface, decay, if it occurs, will start in the portion of the enamel that is in near contact with the neighboring tooth.

Therefore, we see again in this that the nature or perfection of the enamel is in no wise an element in the localization of decay on the smooth surfaces of the teeth. These unusual forms, and apparently unusual positions, of beginning caries are always

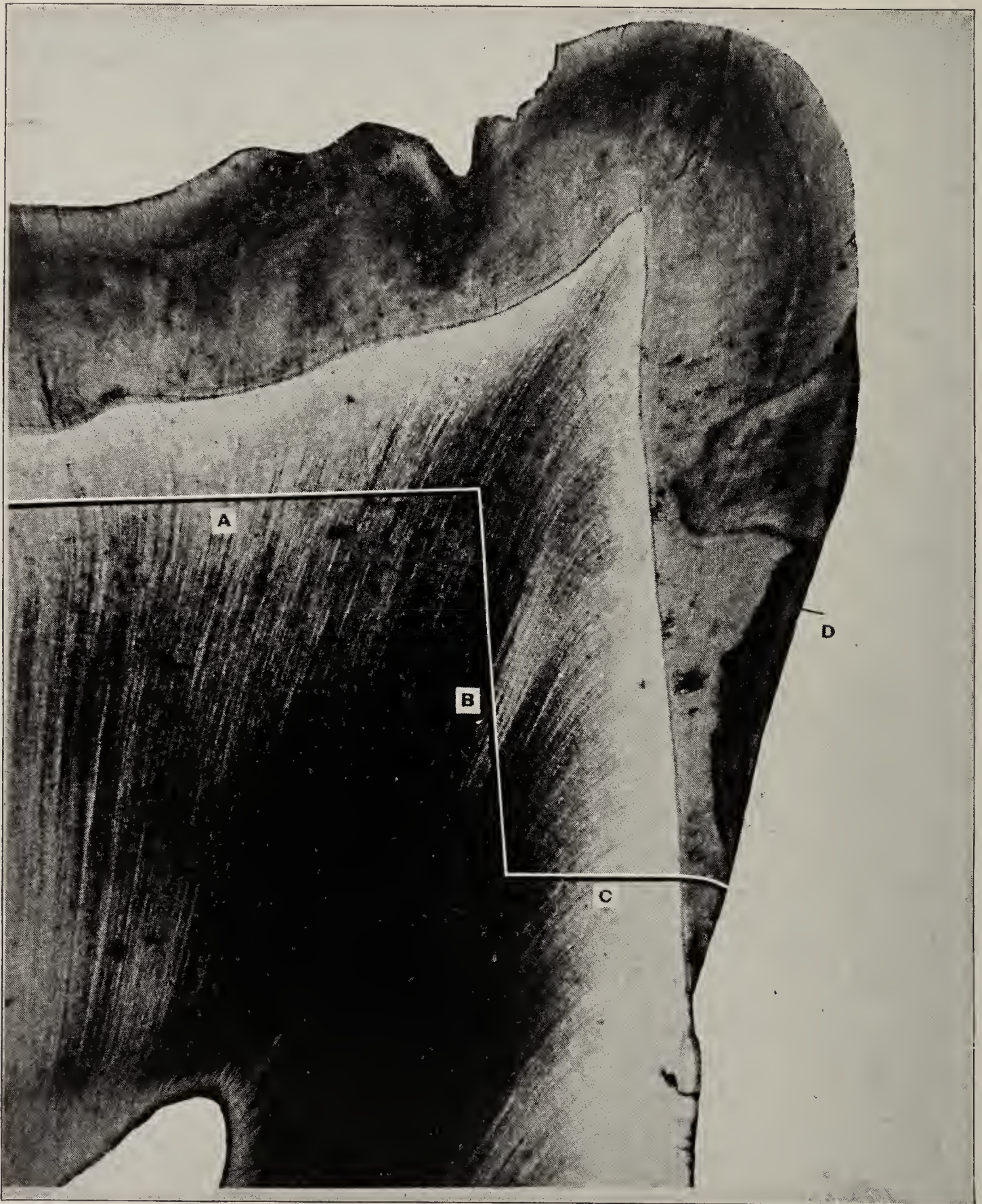


FIG. 209. A photomicrograph of caries of the enamel on the proximal surface of a molar, with an extension gingivally. The decay from the original point of beginning has penetrated the enamel almost to the dento-enamel junction. The extension gingivally is partially separated from the principal decay. In the preparation of a cavity for a restoration in such a case, it would be an error to cut the gingival wall at D. The true cavity lines would place the gingival wall at the line C; the axial wall on the line B; the occlusal wall of the step on the line A.

traceable to some condition that serves to invite lodgment and to protect colonies of microorganisms. In the examination of many teeth, such unusual positions of beginning decay are not very rare.

SECONDARY EXTENSIONS GINGIVALLY OF PROXIMAL DECAYS.

ILLUSTRATION: FIGURE 209.

In proximal surfaces another condition arises frequently that deserves especial attention on account of its great clinical importance. Figure 209 shows a carious area of enamel that has not yet reached the dentin. In this case, a secondary beginning of caries of the enamel has occurred toward the gingival at D, extending almost to the cemental line. Because of roughening of the surfaces or because of the falling away of enamel rods in one or both of the approximating teeth, the food, instead of gliding out laterally in the normal way, will be held, and will be forced more and more onto the gum tissue as other food is forced upon it. In this way the interproximal gum tissue will be absorbed and a pocket will be formed between the teeth that will be well enclosed by the festoons of the gum to the buccal and lingual, and coverings of debris will protect it from washings by the saliva. Acid fermentation will become established in this pocket. The acid formed by this fermentation will be in contact with the surface of the enamel and its calcium salts will be dissolved.

Conditions which cause the food to lodge become a cause of the wide secondary extension of the carious area toward the cemental line, which creates a serious complication that is too often overlooked at a time when it might be easily remedied. During the preparation of the cavity, such an extension of decay will show a white area of more or less thickness on the cavo-surface angle of the gingival wall, while the remainder of the enamel wall will be hard and firm. In this case, if the original decay near to the point of contact had extended into the dentin and the cavity had been discovered by the breaking away of the enamel at a time when the secondary extension of decay gingivally, as shown, was at its present stage, which often occurs, it would have been easy to overlook this extension and prepare the cavity with its gingival wall cut at the line D, instead of cutting the cavity to the line C. Such an error as cutting the gingival wall at D would inevitably have resulted in disaster within a short time. In practice, the only way in which to make a restoration that will not soon be undermined at the gingival wall is to continue the extension until all appearance of this secondary caries of the enamel has been removed. The perfect enamel will then show the usual solid vitreous appearance at the cavo-surface angle of the gingival wall. Then the contact point must be so formed and the restoration so finished as to prevent the leakage of food into the interproximal space later on. Afterward, the regrowth of the interproximal gum tissue should be en-

couraged by special attention to local cleanliness and the case should be kept under observation with the expectation that it will refill the interproximal space.

Any condition which allows food to leak into the interproximal space, be held and forced onto the gum tissue, will cause the absorption of the gum tissue and result in the formation of a pocket. In time, one of two things is certain to happen: (1) acid fermentation will become established in the pocket and decay will occur farther toward the cemental line, or (2) putrefactive decomposition will become established, and finally disease of the peridental membrane will result. Careful clinical study has shown conclusively that much the greater number of the decays met with in practice that extend far toward the cemental line, or past it into the cementum, have occurred in this way. It is true, however, that a considerable proportion of these have occurred in what may be called the more normal way by the spread of decay along the dento-enamel junction and backward decay of the enamel. This latter can occur only in the badly neglected cases.

The crowding of food between the teeth after the placing of so-called contour restorations in the earlier days of cohesive gold was the cause of widespread loss of restorations by undermining by decay at the gingival margin. The former employment of the separating file as it had been developed in the use of non-cohesive gold was continued for a time in the finishing of cohesive gold restorations. Flat contacts were made and the forms of proximal surfaces were left otherwise in imperfect form. There had not been that close study of tooth forms which enabled practitioners to copy them with accuracy, or to appreciate the correctness of forms of interproximal contacts; neither did they have suitable instruments. It is only by the study of the best natural forms of interproximal contacts and their function in the protection of the septal tissues, the copying of these in the shaping of proximal surfaces of restorations, which are made to restore the full mesio-distal breadth of the teeth, that this difficulty in the treatment of caries of proximal surfaces is being overcome. Decays recurring from these causes are especially difficult of treatment, often requiring the removal of a restoration previously made, in order to reach them from the occlusal surface. They are also complicated with difficulties in getting the rubber dam far enough to the gingival to protect them from moisture.

Therefore, in practice, a complaint of pain being produced by food lodgments, or of food being held between the teeth in the chewing of meats or other stringy foods, should receive immediate attention, the cause found and the condition remedied. It may occur from a number of causes besides the beginning of caries, and will occasionally be found in one, two or more teeth, in mouths in which no caries has previously occurred. The proximal contacts may be bad from faulty forms of the teeth themselves, they may

have become bad from movements of the teeth after extractions, the contacts may have become flattened by excessive proximal wear, but oftenest of all, they have become bad because of beginning of proximal decay. No service will elicit keener expressions of appreciation than the correction of faulty contacts. It constitutes one of the real opportunities in preventive service.

INJURIES BY EXCESSIVE PROXIMAL WEAR.

ILLUSTRATIONS: FIGURES 210-214.

The clinical consideration of caries of the proximal surfaces of the bicuspid and molars should not be passed without more special mention of the injuries that result from excessive proximal wear and the flattening of the contact points from this cause; though it will be again presented from the technical view in discussing restorations. The general principles governing the lodgment of food debris between the teeth have been mentioned, with excessive proximal wear of the contact points as one of the causes.

A certain indefinite amount of wear of the mutual points of proximal contact between the teeth as they stand in the arch, must be regarded as normal. Almost any tooth extracted after the age of twenty-five or thirty years will show a facet of wear on its point of contact with its fellow. A number of measurements of these give an average of a loss of about one centimeter in the length of the arch from this cause when measured on the labial and buccal surfaces of the teeth around the arch from the mesio-buccal cusp on one third molar to the other at the age of forty years. This wear increases as the person grows older. When this wear is fairly even in its distribution among the several teeth, it can not be regarded as abnormal, nor is it a cause of material injury. Such wear does not loosen the normal pressure of the contact of tooth with tooth as they stand in the arch in any degree. In the balance of forces which confine the teeth in normal form and occlusion in the arch, there is a moderate but continuous pressure exerted to hold them firmly one against the other, which, when conditions remain normal, continues through life. This is much more than sufficient to take up any loss of length of the arch around its curve that may be occasioned by the wear of the contact points. This is often shown by the quickness with which the teeth anywhere in the arch will close together when a contact point has been lost by reason of caries or the reduction of an intervening space where a tooth has been removed. This wear is produced by the slight movement of the teeth in their alveoli allowed by the periodontal membrane. It is not equal in all parts of the mouth, but is greatest among those teeth which do the heavier work in chewing food, especially the second bicuspid and the first and second molars. In these teeth it is frequently excessive. The five first molars, photographs of which illustrate



FIG. 210.



FIG. 211.



FIG. 212.



FIG. 213.

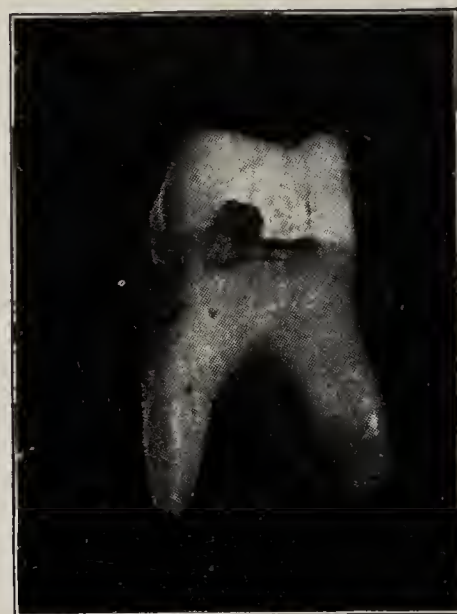


FIG. 214.

FIGS. 210-214. This group of five teeth, all of which were extracted by the author because of neglected disease of the peridental membranes, caused by the crowding of food between the proximal surfaces, flattened by interproximal wear. These should have been protected at the proper time by building prominent contacts for the cure of the difficulty. Figures 213, 214, illustrate the position in which decay most usually occurs in these cases.

this subject, were each removed by the author because of injury done to their peridental membranes by the food which was held by the flattened surfaces and crowded against the interproximal gum tissue. The flat facets shown in the photographs exhibit the amount of proximal wear that may be expected to occur frequently in persons fifty to sixty years old, who have made good use of their teeth. Many cases may be observed that have become worn as much as these, or nearly so, in which no special harm has resulted, and they require no attention. But in a certain number of these, food that is unusually tough and stringy will occasionally be forced between the teeth and not be removed. At subsequent meals more will be forced in, until finally the pressure of the con-

tact will be loosened and remain so. Then trouble has begun in earnest, which, if not relieved promptly, will certainly result in disaster. Within the author's observation, certain persons have manifested a remarkable unconcern as to this condition, claiming that they had never experienced any uneasiness whatever, even when large amounts of gum tissue had been destroyed by the pressure of food debris. Such cases are often hopeless when first seen. But when the dentist discovers such cases in time to act successfully, he should express the necessity for treatment by proceeding at once to do that which is necessary. Such a course will save his patient from the loss of the teeth concerned. Others, and much the greater number, are in constant trouble from the beginning of the lodgments and gladly accept anything that promises relief. The number of persons who have complained that they have been unable to obtain relief when applying to their dentist indicates that the body of the profession has been slow to realize the necessity for treatment, or to see the way to make it successful. As said in the previous article, one of two things is sure to result if this continues: (1) acid fermentation will become established in the pocket formed between the teeth as a result of the absorption of the interproximal gum tissue, by the pressure of the accumulations, and caries beginning near the cemental line, as shown in Figures 213, 214, will result; or (2), putrefactive decomposition will occur, resulting in disease of the peridental membrane. In Figure 210 a considerable absorption is shown on the side of the mesial root of the lower molar, which seems to have been caused by the continued irritation of the peridental membrane, a thing that has been observed in a considerable number of such cases.

The common habit of dentists of discarding extracted teeth without examination of the condition of their roots, is accountable for the slowness of the development of our knowledge of the effect of pathological conditions upon the peridental membranes and the hard tissues which they invest. In this illustration, Figure 210, the surface flattened by wear is very broad. In Figure 211 a decay of the enamel had begun near the contact point but had ceased to progress because of some favorable change of conditions. This has become blackened and the facet of wear has later spread over it. In Figure 212 the enamel had been worn entirely through, exposing the dentin. The location of the decays that have begun in Figures 213, 214, is typical of the beginnings of decay in these conditions and speak for themselves as to the difficulty of treatment. From every point of view cases of this class call for immediate, careful consideration and treatment in the very early stages of their progress, or just so soon as it is noticed that the interproximal soft tissues are being injured. Only one thing promises relief, and, fortunately, has proven very effective when carefully done. This is to cut a cavity in one of the worn teeth that shall

fully include the worn area, make a good and sufficient separation of the teeth and build out a prominent contact that will hold the surfaces sufficiently apart and prevent further leakage of food into the interproximal space. This treatment is given in detail in discussing the technical procedures in making restorations.

PROXIMAL SURFACE DECAYS IN INCISORS AND CUSPIDS.

ILLUSTRATIONS: FIGURES 177-183; 215-219.

PRINCIPAL CLINICAL FEATURES: (1.) The V-shaped form of the proximal surfaces. (2.) The necessity that cavities be approached through the labial or lingual embrasures, differing from the approach through the occlusal surface in the molars and

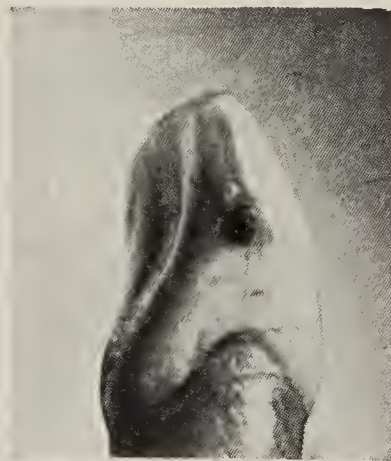


FIG. 215.



FIG. 216.

FIG. 215. A blackened spot on the mesial surface of a central incisor caused by a beginning decay of the enamel which was stopped by a change of conditions, and afterward became very dark. It shows the most common position of beginning of decay in these surfaces.

FIG. 216. An open cavity in the mesial surface of a central incisor with a superficial extension of decay running away from it, toward the linguo-gingival angle of the surface. A similar superficial whitening leads away to the labio-gingival angle, which was lost in the high light in photographing.

bicuspid. (3.) The curvature of the surface at the usual point of initial attack carries extensions of decay along the dento-enamel junction quickly to the undermining of the lingual or labial enamel plates, or often both. (4.) The frequent danger of the spread of decay incisally along the dento-enamel junction, destroying the support of the incisal angle. (5.) The tendency to spreading of caries to the linguo-gingival and labio-gingival angles of the surface, either before or after restorations have been made. (6.) The triangular forms of prepared cavities, with extensions at the labio-gingival and linguo-gingival angles only, instead of the square-cut cavities in proximal surfaces of the bicuspid and molars. (7.) The necessity for preparing incisal anchorages of a form not used elsewhere. (8.) The greater necessity for esthetic considerations in all parts of the treatment, and especially in the preservation of the stronger parts of undermined labial enamel. (9.) The great danger of injury to the attachment of the soft tis-

sues to the tooth at the crest of the arch of the cemental line on the mesial and distal surfaces in the use of ligatures.

The two groups of Figures, 177-183; 215-219, taken together, present a progression from the very early beginnings of caries in the enamel in proximal surfaces of incisors and cuspids to a very considerable invasion of the dentin. They give a fair view of the usual conditions found, including the place of beginning and the manner and direction of the invasion. Particular attention should be given to the arch of the gingival line as it passes from labial to lingual across the proximal surfaces. This is

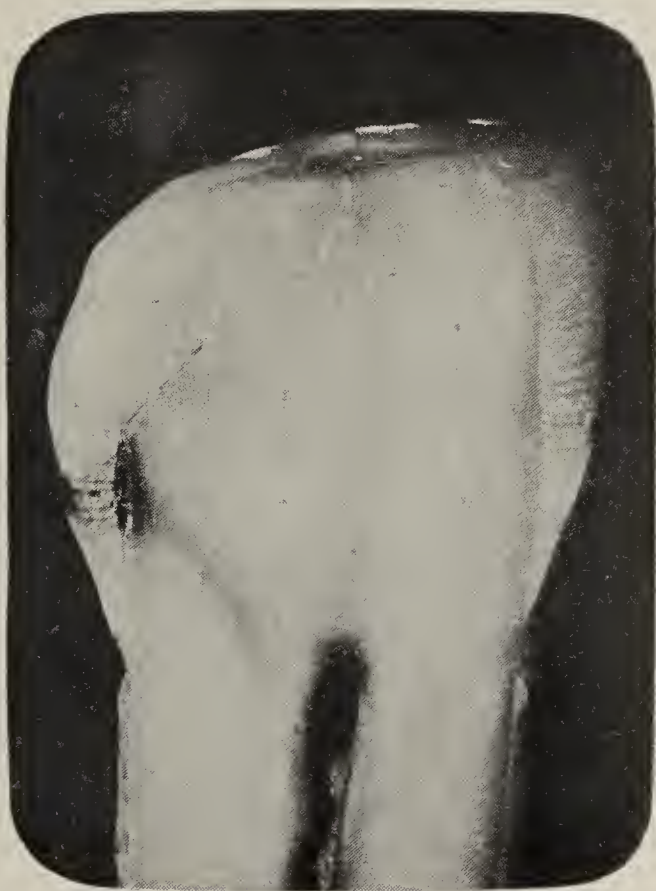


FIG. 217.

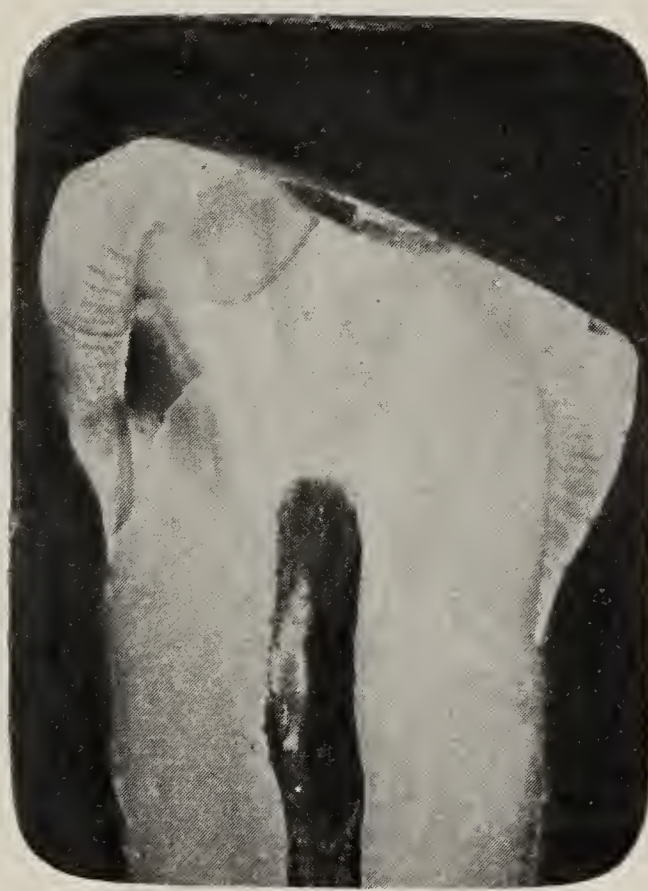


FIG. 218.

FIG. 217. A beginning decay in the distal surface of a lateral incisor from which the enamel rods are falling away and progress of decay has begun in the dentin. A delicate hyaline area stretches away toward the pulp chamber.

FIG. 218. A decay in a worn cuspid that has made greater progress. This picture is liable to give a false impression for the reason that the cut is a little to the side of the central area and shows the enamel penetrated with the enamel rods still in position, when, in fact, the cavity was open through the enamel.

well shown in Figures 215, 216, which exhibit plainly the danger of serious damage to the attachment of the soft tissue to the tooth at the crest of the arch of the cemental line by tying ligatures tightly and forcing them to the cemental line on the labial and lingual surfaces. This danger is found particularly with the incisors and cuspids, and great damage is frequently done by inattention to this point.

Figure 215 shows particularly well the most common position of beginning caries on the mesial surfaces of the incisors. It is sometimes a little closer to the incisal angle and sometimes a little farther away, though it does not often vary greatly from

the point shown. The spot shown is a beginning decay which had penetrated the enamel but little apparently, and, having been stopped by a change of conditions, became very dark. Figure 216 was intended to show the broad spreading of caries which sometimes occurs on the proximal surfaces of these teeth. This is plainly shown on the lingual in the rounded tongue of superficial decay extending away from the dark, open cavity toward the linguo-gingival angle of the surface. A somewhat similar extension toward the labio-gingival angle was apparent, but the high light in the photograph has hidden that point. Such extensions as that seen upon the lingual in this photograph are particularly liable to occur in very susceptible persons after restorations have been made, unless the angles of cavities have been extended to include the susceptible area. Otherwise, this case presents a wide-open cavity in which the undermined enamel has broken away most toward the lingual surface. The penetration of dentin and its

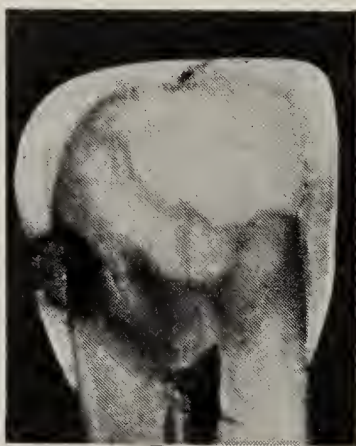


FIG. 219. A more considerable decay in the distal surface of a central incisor in which the undermining of the enamel is less than usual. In such a case the labial or lingual plate of the enamel may be found undermined.

direction of progress is progressively shown in Figures 217, 218, 219. In the first of these, the enamel rods have fallen out, and the spreading of decay along the dento-enamel junction is in progress. The faint hyaline zone is seen reaching almost to the pulp chamber. This decay is rather nearer the cemental line than usual, because the strong rounding of the distal surface inciso-gingivally placed the contact point unusually far from the incisal. We see in this that the form of the particular tooth plays its rôle in the particular locality of the point of attack in the enamel by caries. The next photograph, showing decay in the mesial surface of a cuspid, Figure 218, gives a false impression in that it shows the enamel rods in position, while, in fact, the cut is slightly to one side of a small area from which they had fallen out, admitting microorganisms to the dentin. The same spreading along the dento-enamel junction is present, though in less degree than the average of cases. An examination of this case will show the liability of extension along the dento-enamel junction undermining

the incisal angle before an exposure of the pulp would occur, a thing that frequently happens to the incisors whenever there is a lack of watchfulness of the progress of decay. This was not a young tooth, as shown by the wear of the cusp, which has exposed an area of dentin. A trace of a hyaline zone is seen streaking away to the pulp chamber from that as well. These shadows occur in abrasions the same as in caries, but usually are not so prominent. Whatever else these zones of shadow may be, they express an injury to the dentinal fibrils. A still more extended invasion of dentin is photographed in Figure 219. This is not an inordinately large cavity, but one that is easily managed in making a restoration. However, even in this, one is liable to find the labial or lingual enamel plates considerably undermined by extensions of decay along the dento-enamel junction. It should be noted particularly that many of the incisors are thin labio-lingually at the point first invaded by decay, and a comparatively moderate extension along the dento-enamel junction may cause such injury to the labial enamel plate as to make a decisive esthetic blemish. This can be avoided only by careful watchfulness over these teeth to see that caries in them receives early attention.

Material for the illustration of this class of decays is exceedingly difficult to obtain and much dependent upon accident. This is exhibited in Figure 178. In this, decay had practically destroyed the central incisor by exposure of the pulp before the apex of the root had closed sufficiently to permit of a root filling. The case exhibits in a striking manner the breadth mesio-distally of the pulp chamber at this tender age of the child, the proximity to the pulp of the usual points of the beginning of caries, the small amount of dentin through which decay must penetrate to expose the pulp, and strongly suggests the watchfulness that should be had over such teeth in families highly susceptible to caries.

GINGIVAL THIRD DECAYS IN LABIAL AND BUCCAL SURFACES.

ILLUSTRATIONS: FIGURES 220-224.

PRINCIPAL CLINICAL FEATURES: (1.) The earliest beginning of decay is a line of whitening of the enamel running mesio-distally near the margin of the gum in the middle third of the surface mesio-distally. (2.) The spreading of the decay on the surface of the enamel is usually confined closely to extensions mesially and distally toward the angles of the tooth, following the curve of the border of the gum. (3.) In cases of neglect of cleanliness, and especially in neglect of the use of the teeth in chewing food, there may be extensions occlusally and also across the angles of the teeth to connect with proximal decays on the mesial and distal surfaces. (4.) In many cases of this class of caries the disposition is seen to spread quickly from tooth to tooth, or to attack a number of teeth at the same time. (5.) Yields to prophylactic treat-

ment by the patient, when properly instructed, more readily than any other class of decay. (6.) Protection from recurrence of decay after making restorations is had only by extension of prepared cavities nearly to the angles of the teeth in the ordinary cases. (7.) Attacks fewer persons than other classes of caries, but is often very destructive when a beginning is once made. (8.) The most general rule is that gingival third decays occur later in the life of the person than the other classes, but their occurrence in early youth is not very infrequent.

Gingival third decays in the labial or buccal surfaces, or in both together, have been much dreaded by dentists because of persistent recurrence to the mesial and the distal of the margins of



FIG. 220.

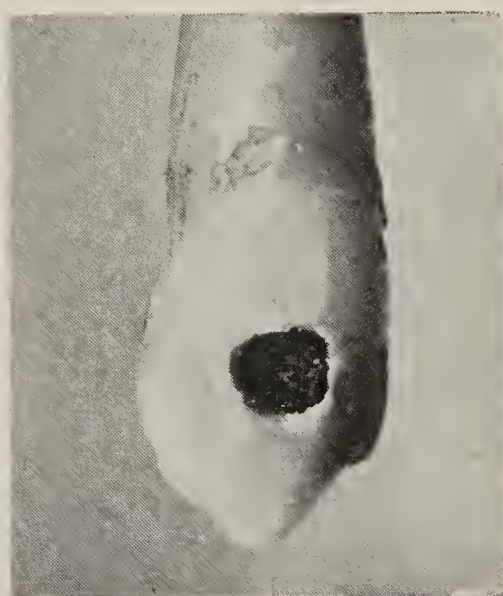


FIG. 221.



FIG. 222.

FIG. 220. A photograph of a cuspid with a decay across its labial surface. There is a decayed area running across the surface mesio-distally that has penetrated the enamel and is making progress in the dentin. Undermined enamel has been breaking away, leaving more or less jagged margins. The beginning of this decay occurred when the free margin of the gum was about at the gingival margin of this carious area, and covered the portion of the tooth to the gingival of it. As the tooth protruded farther through the gums, more of the enamel became exposed and the conditions producing decay continuing or recurring, another band of whitened enamel—beginning decay—has occurred to the gingival of the first.

FIG. 221. Another cuspid, similar to that in Figure 220, in which a decay, beginning in the enamel when the tooth was still half covered with gum tissue, has become fixed in the dentin and later produced a round opening by the breaking away of undermined enamel. When the tooth had protruded farther through the gum and the conditions causing the beginning of decay in the enamel having passed away, this appeared much removed from the free border of the gum.

FIG. 222. A molar showing two beginning points of decay on the buccal surface. A whitened line running mesio-distally was apparent on this tooth, but the engraver failed to show it.

restorations, or to the gingival when these have been made for young people. This difficulty has arisen from a failure to study the clinical characters and conditions of occurrence closely enough to direct properly the treatment for its eradication and cure. If the conditions which have given rise to the beginning of the decay are not materially modified by prophylactic measures, or spontaneous cessation, the disposition to spread mesially and distally is one of its most persistent characters. Nothing less than the

extension of cavities very nearly to the angles of the teeth attacked, will be effective.

Figure 220 is a photograph of a cuspid with a gingival third decay which began before the tooth fully reached its position in the arch, or when the crown had yet more than one-third of its length covered by the margin of the gum. Decay spread rapidly across the surface mesially and distally from the beginning point and became established in the dentin. In the process of growth, more of the crown of the tooth was uncovered to the gingival of this decay, the conditions which caused the first beginning remaining or recurring. The result is another whitened band of enamel, a new beginning of decay, to the gingival of the first. This is what may be expected to occur when such decays are treated by restorations, when they occur in young persons, unless the margin of the gum is pushed well away and the cavity margin extended so far to the gingival as to include the new area of liability that must become exposed. In early youth the sufficient persistence of attention to prophylactic measures for the prevention of such extensions is not likely to be so well kept up as to be a very safe protection.

The most essential condition for the occurrence of decay of this class is a saliva favoring certain kinds of deposits upon the teeth which will cover attached colonies of microorganisms in such a way as to protect the acids formed from free dissipation in the general oral secretions. This may be such a condition as will enable microorganisms to protect themselves by the formation of gelatinous plaques, or possibly by other kinds of deposits from the saliva that will form a membrane-like covering that will afford a sufficient protection. It is a notable fact that where any kind of deposits are found upon the teeth, they are first to be seen on the buccal surfaces at the points where gingival third decays begin. It is not any and every kind of aggregation of deposit on the surfaces of the teeth that gives rise to decay. Indeed, some forms of deposit seem to prohibit decay instead of causing it. Some mouths that are very unclean have no caries, and in some cases where caries has been in progress it seems to have been stopped by extreme lack of cleanliness in the same way as decay not infrequently is stopped in certain cavities by the establishment of putrefactive decomposition in their interior, as has been mentioned. Neither will loose aggregations of saprophytic microorganisms, or of leptothrix buccalis, or the leptothrix Vignon, or other harmless varieties which may form thick masses over the teeth, through which water will run as freely as through a sieve, have any influence favoring the production of caries. *The covering must be of a kind that will protect from free washings by the fluids of the mouth, and in and under which the microorganisms will grow and produce acid fermentation.* This may be so thin and so transparent that the teeth may appear clean and yet afford an effective protection against the too free dissipation of the acid products of

fermentation. It has become evident enough from clinical observation that the conditions which favor the formation of these plaques is one that is liable to be intermittent. It comes and goes. Decays of the gingival third of buccal and labial surfaces are especially liable to start, to stop, and to start again. Their exposed situation renders them more sensitive to fluctuations of conditions than decays situated in protected localities. This is said of decays of the enamel in which enamel rods have not fallen away, exposing the dentin to invasion by microorganisms. Once caries is implanted within the dentin, it will most generally persist, even when there is complete immunity to the beginnings of caries on the enamel.

Figure 221 shows a decay, situated centrally in the labial surface of a cuspid, which has progressed in the dentin, undermining the enamel, which, by breakage, has now formed a round opening. At a time when this tooth was taking its place in the arch and was uncovered by the gum tissue, only to about that point, a decay penetrated the enamel and became seated in the dentin. The particular form of the beginning in the enamel is now lost by breakage from backward decay, but observations of such cases indicate that it was in a line across the central portion of the present dark area. As the tooth protruded farther through the gum, the conditions causing the beginning of decay in the enamel passed away and did not return, but the decay established in the dentin continued. Had the original beginning failed, ever so little, to penetrate the enamel and admit microorganisms to the dentin, the progress would have ceased entirely. Later, a blackened blemish of the enamel would have remained to show where decay had begun.

The gingival third decays in the buccal surfaces of the bicuspids and molars are not materially different from labial surface decays. They exhibit similar characters in both their beginning and in their disposition to spread along the gum margin mesially and distally. In the photograph of the upper molar, Figure 222, two considerable areas of loss of enamel rods appear in an area of rather faint whitening stretching across the buccal surface. Figure 223 shows the extensive injury that sometimes befalls these teeth when caries is allowed to go on unchecked by any sort of care. For this illustration, the cementum has been tinged with a selective anilin stain to show the cemental line distinctly in order to bring prominently into view that portion of the enamel covered by the border of the gum, illustrating its protection from the beginnings of caries.

Figure 224, a photograph of an upper third molar, presents an anomalous condition. In coming into position it deviated backward and to the buccal from the normal, and seemed to have stood

for some time with but a part of its crown through the gum. When removed, the whole of its exposed surface was whitened by beginning decay.



FIG. 223.



FIG. 224.

FIG. 223. Photograph of a lower molar tooth, in which caries proceeding from a buccal surface decay and a mesial surface decay have met across the angle. This shows the wide range of the decay of the buccal surface. This meets the mesial surface decay across the angle of the tooth. This picture also shows the breaking away of the undermined enamel of the buccal surface to advantage. The cementum of this tooth was stained selectively with an anilin dye to bring the cemental line into prominence, showing the influence of the free border of the gum in protecting the enamel from beginning of caries.

FIG. 224. A photograph of an upper first molar that was misplaced toward the cheek and but partially erupted. When removed, decay of the enamel had begun over all of the exposed surface.

SPREADING OF DECAY AROUND THE TEETH.

ILLUSTRATIONS: FIGURES 225-228.

In what has been written thus far of dental caries, the idea has been developed that, when decay occurs on proximal surfaces, the tendency to superficial spreading is from the starting point both buccally and lingually toward the angles of the teeth. Also, that, when caries begins on the buccal surfaces, the tendency is to spread mesially and distally from the place of beginning toward the angles of the teeth. This is true of caries in these positions in all of the teeth, but more especially of the bicuspid and molars. A fewer number occur in the front teeth as well. It has also been stated that in a few instances under specially unfavorable conditions this decay crosses the angles of the teeth and the proximal and buccal decays join each other. This crossing of the angles of the teeth is the rarest of all of the spreading. It then requires only that decay shall also occur similarly on the lingual surfaces in order to complete the circle of the tooth. This, though much more rare, occurs also. This appears in the case of the lower second molar tooth, four photographs of which are shown in Figures 225-228, inclusive. Figure 225 shows the mesial surface with a broad,

whitened area of carious enamel stretching from angle to angle, in which the enamel rods have begun to fall away at one point only. Figure 226 is a photograph of the buccal surface with the whitened area also stretching from angle to angle, the shadow obscuring a small part at the mesial angle. The distal surface, Figure 227,



FIG. 225.



FIG. 226.



FIG. 227.



FIG. 228.

FIGS. 225-228, inclusive. Photographs of a lower first molar with beginning decay completely encircling the crown, following closely the free margin of the gum in every part. Figure 225, the mesial surface; Figure 226, the buccal surface; Figure 227, the distal surface; Figure 228, the lingual surface.

shows dark, but the decay is apparent, and on the buccal surface, as seen in this photograph, the whitened line of carious enamel is seen to advantage as it rounds to the occlusal following the margin of the gum to join the distal surface decay. Finally, the whitened decay on the lingual surface, Figure 228, is seen streaking away from the decay in the mesial surface in a curved line, which is lost in shadow as it approaches the distal angle, completing the circle of the tooth.

The pictures of this series are the final illustrations for the explanation of the tendencies to spreading superficially on the surface of the enamel and the direction of that spreading as one of the principal clinical features of caries of the teeth, which every dentist should fully understand and appreciate as his guide in the preparation of cavities for the prevention of recurrence of decay about the margins of restorations. Every one should understand distinctly that the spreading *on the surface of the enamel* is a thing entirely different and apart from spreading along the dento-enamel junction and destruction of the enamel by backward decay, together with the general invasion of and destruction of dentin by caries. This latter spreads in every direction from the point of penetration of the enamel, having no respect whatever for any particular surface or any of the angles of the teeth. The spreading along the dento-enamel junction this way or that has no clinical significance in connection with the recurrence of caries about restorations. This invasion may produce broad cavities, cavities of awkward shapes, may weaken the tooth by the destruction of dentin, and, in these and other ways, has its special points of clinical importance. But this is all secondary, occurring only after the enamel is broken. The clinical importance of a full appreciation of the superficial spreading of caries on the enamel has to do especially with the prognosis, with the probable future of every case individually, and the rational management of the teeth of each person under our care. On the details of this management, the success or failure will depend more than all else, supposing always that the details of manipulation, as this may be planned, be skillfully executed. Together with all of this, judgment must be stimulated and quickened by a careful study of the conditions of immunity and susceptibility to dental caries.

CARIES IN CASES OF RECESSION OF THE GUMS.

ILLUSTRATION: FIGURE 229.

Decay occurs in the teeth of elderly people about the gum margins when these have become very short. The decay is apt to invade the cementum soon after its beginning and for this reason is unusually difficult to treat successfully. In many cases it begins in the cementum after more or less recession of the gum. See Figure 229, a section of a bicuspid which had been in service for many years without being affected with caries. The decay began in the surface of the cementum, a little below the cemento-enamel junction, following recession of the gum. Some cases are complicated by exposure of the pulp. These decays are not different in the pathological sense from other classes of caries of the teeth. It is only modified in form by the general conditions under which it occurs. Generally it will be found that such persons have become negligent in their habits of cleanliness. The free margins of the gums have practically disappeared and lodgments of debris are

plentiful about the necks of the teeth. Under these circumstances the resulting decay takes forms that are peculiar to the local conditions. However, these decays occasionally occur in mouths which are kept very clean. The treatment of cases of this type is given elsewhere.



FIG. 229. Decay which began on the surface of the cementum, subsequent to recession of the gum.

UTILITY OF STUDIES OF DENTAL CARIES.

When Dr. Miller made out the life history of the microörganisms which cause caries of the teeth and fully determined their action in carious dentin, it was hoped by many that this would be a guide to treatment that would become of great advantage in the practice of dentistry. The profession did not appreciate the wide difference between caries of dentin and caries of enamel, and did not realize the greater importance of an understanding of caries of enamel — which is necessarily the first tissue involved in dental caries — in the practical application of restorative operations for the control of caries. The importance of this factor in the practical use of Dr. Miller's findings in the treatment of dental caries has come very slowly to the minds of men; but it must now be seen that these findings have been the basis of advancement in the study of caries of enamel, which has directed the formulation of our principles of treatment. The interpretation which held that the teeth decayed because of inherent weakness in the teeth themselves, or from variations in their calcium salts, seems to have been responsible for much of this delay. The profession has been

very slow to understand that caries of the enamel is the principal factor to be considered in any treatment that may be instituted for the control of dental caries. The growth of microorganisms in the saliva cannot be prevented; therefore our attention must be turned to the question of preventing or limiting the injuries they do to the teeth, while our knowledge of the etiology of caries is gradually perfected.

Caries of enamel is the initial lesion and always occurs as the beginning process of dental caries. When the enamel has been penetrated and the carious process becomes established in the dentin, the only logical method of treatment is by its eradication by excavation and placing a restoration. Caries of enamel, however, always begins upon the outside of the tooth, or in pits and fissures in the surface, and methods of prevention and control have both been more effectively applied in recent years. Pit and fissure decays have been prevented in many mouths by proper brushing, they have been almost completely controlled for a high percentage of patients by the restorative treatment herein outlined.

Experience in directing the prophylactic treatment of labial and buccal surfaces shows plainly that decay of these can generally be perfectly controlled by the use of the tooth brush and plain water by the patient, whenever the habit is sufficiently formed that it will not be neglected. Restorations in gingival third positions in these surfaces are rarely required to-day in practices in which good patient co-operation has been established.

Proximal decays can not be prevented by the most faithful and thorough mouth hygiene technic, nor by the most frequent cleaning by the dentist that has been found practicable. The best possible combination of the two methods has been ineffective in preventing the occurrence of decay of these surfaces in more than a small percentage of persons. There can be no question but that the application of these methods is helpful in retarding the progress of caries in susceptible individuals, but a broad study of the situation is convincing that they are aids only and are not to be considered as reliable preventive measures.

At the present time the application of ammoniated silver nitrate to these surfaces at regular intervals after the teeth erupt gives promise of more effective control, with the possibility of immunizing the areas of liability. More time will be required to demonstrate the value of this method. The technic is simple and the reports available after its use over periods of five years or more by a considerable number of practitioners, should encourage the more general adoption of the application of silver nitrate as a routine treatment.

The logical application of extension for prevention in cavity preparation, and the selection of materials of proved merit as to their physical properties for restorations have greatly improved

the control of proximal decays. The change from the purely mechanical treatment to the scientific method, based on knowledge of the pathology of caries, is taking place very slowly, as was to be expected. It requires the passing of two or three generations, to make so radical a change in thought and procedure generally effective. To learn to use instruments deftly and to make an excellent restoration from the mechanical standpoint is essential, but this is not enough. The planning of the restoration must be such as to adapt it, not only to the cure of the particular decay, but to prevent the recurrence of caries in the future.

To do this wisely requires a closer study of the beginnings of dental caries in the enamel than has yet been made by the general body of the profession. When this has been accomplished and the knowledge of these processes has become generally diffused, the treatment of dental caries will be far more successful than it is to-day. The systematized treatment for prevention of caries of the teeth, can not be wisely done without better understanding of the signs of susceptibility and immunity in each individual. This understanding is necessary as a basis for the more rational application of prevention in every service performed.

The inter-relation of diseases of the dental pulp and of the peridental structures with dental caries is a matter of great importance in the occurrence of chronic mouth infections, and their bearing on the general health. The effects of neglected caries, or of failures to construct proper contours and contacts in making restorations, are as direct in establishing disease of the peridental tissues as is the death of the pulp from failure to control caries. These pathological relations are generally understood, but the practical application of treatment involves the establishment of patient co-operation through years of service and, to be most successful, must begin early in the life of the large majority of persons.

The dentist must realize that his obligation, in performing each operation for a young person, is one that extends throughout the life of the individual, and calls for the highest ideals in professional service. It should be a part of his program to inculcate in the minds of his patients an understanding of the relation of his service to health in later years and thus secure their co-operation to the fullest possible degree.

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